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It contains (1) current information regarding the prevalence and geographic distribution of communicable diseases in the United States, insofar as data are obtainable, and of cholera, plague, smallpox, typhus fever, yellow fever, and other important communicable diseases throughout the world; (2) articles relating to the cause, prevention, and control of disease; (3) other pertinent information regarding sanitation and the conservation of the public health.

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PREVALENCE OF DISEASE

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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

December 29, 1940–January 25, 1941

The accompanying table (table 2) summarizes the prevalence of nine important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the Public Health Reports under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ended January 25, 1941, the number reported for the corresponding period in 1940, and the median number for the years 1936–40.

DISEASES ABOVE MEDIAN PREVALENCE

Influenza.—The number of cases of influenza continued to increase during the first 3 weeks of the 4-week period ended January 25, but decreased considerably during the fourth week. The number of cases reported weekly was as follows: Week ended January 4, 12,905, January 11, 89,828, January 18, 120,006, and January 25, 96,652 cases. The total of 383,630 cases was the highest reported for this period since 1929, when a total of approximately 425,000 cases occurred during this period. During the 1932–33 epidemic there were approximately 144,000 cases reported for this period. The number of cases was almost seven and one-half times the number recorded in 1940 and more than 30 times the 1936–40 median figure for this period.

The current epidemic started in the Mountain and Pacific regions and spread rapidly into the southern areas. For the current period 200,218 cases, or more than 50 percent of the total, were reported from the South Central region, and more than 30 percent from the South Atlantic region (114,502 cases). States in those regions reporting the highest incidence were: Texas (109,820 cases); Kentucky (20,667); Alabama (19,188); Virginia (32,412); South Carolina (28,002); Georgia (25,523); and West Virginia (23,354).

Increases were also noted in the New England and North Central regions, but the incidence in those regions has been low as compared

with the southern and western regions. Maine with approximately 5,000 cases, New Hampshire with about 2,000, and Connecticut with 2,708 cases were mostly responsible for an excess of cases in the New England region; Ohio reported 6,895 of approximately 10,000 cases occurring in the East North Central region, and Kansas reported 8,406 of the 12,169 cases reported from the West North Central region. Further increases may be expected in those regions, as the maximum incidence up to that date was reported during the week ended January 25. In the Mountain and Pacific regions the peak was reached during the week ended December 21 with approximately 27,000 reported cases, while in the South Central regions the maximum weekly incidence was reported during the week ended January 11, and it is probable that the week ended December 25 will be the peak week in the South Atlantic region.

Mortality from all causes for the total number of cities reporting shows some excess during this period, the rate for January being 13.7 per 1,000 compared with an average rate for the years 1938–40 of 13.1 per 1,000. This excess in mortality from all causes is a reflection of the current influenza epidemic. The death rate for pneumonia as reported to the Public Health Service is below the average of the previous 3 years for January, while the death rate for influenza is well above the average rate for the 3 previous years.

Mortality from all causes is further analyzed in table 1, where rates are shown for the 4 weeks of January for nine geographic sections of the United States. In the Pacific section where the current influenza epidemic first appeared, mortality from all causes was somewhat above normal during December (not shown in the table), and has continued slightly above normal during January. In the Mountain and the two West Central sections, mortality from all causes was definitely above normal during the first week in January and has continued to be slightly above normal in the later weeks of January. In the East South Central section, mortality from all causes was slightly above normal during the second and third weeks of January. In the East North Central, Middle Atlantic, and South Atlantic sections there has also been only a slight increase in mortality from all causes, occurring mainly in the last week of January. In the New England States mortality from all causes has been higher than average throughout January, with a marked increase in the rates for the latter half of the month.

Later reports (week ended February 1) indicate a still further decline in the number of cases in practically all sections of the country. For the country as a whole, the cases totaled approximately 73,000, as compared with 96,652 cases for the week ended January 25 and approximately 120,000 for the week ended January 18.

TABLE 1.—*Mortality from all causes in cities in 9 geographic sections of the United States for the 4 weeks of January 1941 compared with an average of the 3 preceding years* [1]

Section	Death rate per 1,000 (annual basis)			
	Jan. 4	Jan. 11	Jan. 18	Jan. 25
All cities reporting:				
1941	12.9	13.7	13.5	14.6
Average, 1938–40	13.1	13.1	12.9	13.2
Pacific:				
1941	13.8	16.6	13.7	15.4
Average, 1938–40	13.4	14.0	14.0	13.6
Mountain:				
1941	21.5	16.7	17.4	15.3
Average, 1938–40	15.3	13.3	13.5	14.6
West North Central:				
1941	15.0	13.1	13.9	14.1
Average, 1938–40	13.4	13.2	12.8	13.6
West South Central:				
1941	20.7	18.3	18.7	17.9
Average, 1938–40	17.8	16.2	16.7	17.3
East South Central:				
1941	14.1	16.3	18.5	17.4
Average, 1938–40	16.1	15.5	14.5	18.6
East North Central:				
1941	11.1	11.8	11.3	12.6
Average, 1938–40	11.6	12.1	11.4	11.2
Middle Atlantic:				
1941	12.3	13.2	13.2	14.4
Average, 1938–40	13.1	12.8	12.9	13.2
South Atlantic:				
1941	13.1	14.9	14.4	16.0
Average, 1938–40	14.6	14.5	14.4	14.0
New England:				
1941	14.8	16.4	19.8	20.8
Average, 1938–40	13.6	15.1	13.5	14.8

[1] Based on data received from the Bureau of the Census.

Measles.—The number of cases (40,419) of measles reported for the current period was more than two and one-half times the number reported for the corresponding period in 1940 and more than twice the 1936–40 median number of cases for this period. Excesses over the seasonal expectancy were reported from the Middle Atlantic, East North Central, and East South Central regions, but in all other regions the incidence was relatively low. In the Middle Atlantic region the number of cases was more than three and one-half times the normal expectancy, and in the East North Central region the number was almost six times the median figure for the period. In the Pacific region, where the disease was unusually prevalent at this time last year, the number of cases was less than one-fourth of last year's incidence, as well as of the 1936–40 median which is represented by the 1940 figure.

Poliomyelitis.—While the incidence of poliomyelitis declined still further during the current period, the number of cases (170) reported was the highest recorded since 1931 when the cases for this period totaled 194. The disease was most prevalent in the Middle and South Atlantic regions and in the North Central regions. In the East North Central region, Wisconsin reported 24 cases, Ohio 14, and Illinois 11 cases; Florida (7 cases) and West Virginia (6 cases) reported the largest

numbers of cases in the South Atlantic region and New York, in the Middle Atlantic region, reported 19 cases. No more than 5 cases were reported from any other State. A further decline in this disease may be expected as the lowest incidence is usually reached during the months of April and May.

Whooping cough.—There were a few more cases of whooping cough than might normally be expected, the cases (16,857) reported for the current period being about 60 percent above last year's figure for this period and almost 10 percent above the 1938–40 median incidence. Each region except the Mountain contributed to the excess incidence.

TABLE 2.—*Number of reported cases of 9 communicable diseases in the United States during the 4-week period Dec. 29, 1940–Jan. 25, 1941, the number for the corresponding period in 1940, and the median number of cases reported for the corresponding period 1936–40*

Division	Current period	1940	5-year median	Current period	1940	5-year median	Current period	194	5-year median
	Diphtheria			Influenza [1]			Measles [2]		
United States	1,220	1,829	2,491	383,630	51,859	12,765	40,419	15,635	18,801
New England	9	53	53	10,051	124	118	2,030	2,583	2,994
Middle Atlantic	180	230	368	1,430	155	155	17,959	1,265	4,863
East North Central	179	351	517	10,012	4,595	621	13,144	2,371	2,371
West North Central	131	119	225	12,169	1,079	919	1,473	1,976	1,976
South Atlantic	250	420	514	114,502	25,134	5,419	2,171	584	2,776
East South Central	109	174	205	52,709	5,278	2,284	1,149	421	421
West South Central	232	297	377	147,509	10,968	3,908	524	883	989
Mountain	58	73	93	21,699	2,383	761	1,061	1,126	1,390
Pacific	72	112	154	13,549	2,143	644	908	4,426	4,426
	Meningococcus meningitis			Poliomyelitis			Scarlet fever		
United States	163	129	377	170	151	85	12,674	16,487	23,617
New England	10	7	11	1	4	1	779	917	1,661
Middle Atlantic	29	33	62	23	13	8	3,314	4,190	4,828
East North Central	18	21	45	60	16	16	4,229	5,490	8,142
West North Central	8	9	28	17	20	7	1,260	1,891	3,676
South Atlantic	34	20	77	28	18	16	1,111	1,287	1,183
East South Central	22	21	66	11	10	10	688	629	620
West South Central	22	3	25	11	14	9	352	533	711
Mountain	4	8	17	7	14	3	342	569	750
Pacific	16	7	16	12	42	15	599	981	1,481
	Smallpox			Typhoid and paratyphoid fever			Whooping cough [2]		
United States	190	320	1,144	312	329	458	16,857	10,405	[3] 15,918
New England	0	0	0	9	19	17	1,551	1,500	1,500
Middle Atlantic	0	0	0	42	57	66	4,481	3,463	3,463
East North Central	64	59	194	46	45	45	3,647	1,859	2,294
West North Central	76	122	450	28	16	39	947	475	475
South Atlantic	3	8	11	48	55	89	2,695	835	2,164
East South Central	5	0	6	26	12	38	466	322	322
West South Central	9	47	47	66	74	101	868	362	469
Mountain	25	64	166	22	33	26	560	713	713
Pacific	8	20	120	25	18	82	1,642	876	876

[1] Mississippi, New York, and Pennsylvania excluded; New York City included.
[2] Mississippi excluded.
[3] Three-year (1938–40) median.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—For the 4 weeks ended January 25 there were 1,220 cases of diphtheria reported, as compared with 1,829, 2,491, and 2,761 cases for the corresponding period in 1940, 1939, and 1938, respectively. The situation was favorable in all sections of the country. In the West North Central region the incidence was slightly higher than during the corresponding period in 1940, but the number of cases was still well below the 1936–40 median incidence for this period. For the country as a whole the number of cases was the lowest on record for this period.

Meningococcus meningitis.—For the current period, there were 163 cases of meningococcus meningitis reported, representing an increase of more than 25 percent over the incidence for the corresponding period in 1940. The incidence was, however, less than 50 percent of the 1936–40 median figure for this period. Regions along the North and South Atlantic Coast and the West South Central and Pacific regions reported excesses during the current period over last year; the Middle Atlantic, East North Central, and Mountain regions reported fewer cases, and in the West North Central and East South Central regions approximately the same incidence was recorded as for last year. In most regions, however, the number of cases was below the preceding 5-year median. This disease has stood at a relatively low level since 1936 when 668 cases were reported for this period; the current incidence represents the first increase over a preceding year's incidence during this period since that year.

Scarlet fever.—For the country as a whole, the incidence (12,674 cases) of scarlet fever for the 4-week period under report was approximately 75 percent of that reported for the corresponding period in 1940 and about 50 percent of the 1936–40 median figure for this period. In the South Atlantic and East South Central regions the incidence stood at about the normal seasonal level; but all other regions reported decreases from last year's figures, as well as very significant declines from the median figures for this period.

Smallpox.—The number of reported cases (190) of smallpox was the lowest on record for this period. Of the total number of cases, Minnesota reported 30, Wisconsin 29, Colorado 23, and Iowa and Michigan 21 each. About two-thirds of the cases were reported from those five States. This disease has been on a steady decline since 1938 when 2,435 cases were reported for the period corresponding to the one under consideration.

Typhoid fever.—The number of cases of typhoid fever reported for the current period was 312, only slightly less than the number reported for the corresponding period in 1940, but about 30 percent lower than the 1936–40 median incidence for this period. In the East North

Central and Mountain regions the incidence was about normal but all other regions reported a relatively low incidence.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ended January 25, based on data received from the Bureau of the Census, was 13.7, as compared with 12.8 in 1940 and an average of 13.1 for the corresponding period in the years 1938–40. By weeks for the current period the rates were 12.9, 13.7, 13.5, and 14.6, respectively. The cause of the increase in the death rate is apparently influenza; further discussion is found under that subject.

THE RESPONSE OF PERITONEAL TISSUE TO INDUSTRIAL DUSTS [1]

By John W. Miller, *Pathologist*, and R. R. Sayers,[2] *Senior Surgeon, United States Public Health Service*

The reaction of the peritoneal tissue to injected dusts has been described in previous reports [3] and attention has been called to the possibility of using the results of such a biological response to predict the pneumoconiosis-producing potentialities of industrial dusts. From time to time, minor modifications in the method of introducing the dusts into the animals have been made to simplify the procedure without altering the results.

As now practiced, the test is briefly as follows: Two cubic centimeters of a 5-percent suspension of air-elutriated (or 325-mesh screened), heat-sterilized dust in sterile, physiological saline solution is injected into the peritoneal cavities of a number of guinea pigs. Animals are killed and examined 14, 45, and 90 days after injection (in earlier experiments at intervals up to 1 year). The nodules produced by the dust on the anterior abdominal walls or in the omentum at the various intervals are compared. The gross appearance is usually sufficient for interpretation of results.

Three general types of reaction are produced by the various dusts. These have been designated as absorptive, proliferative, and inert.

Dusts of the absorptive group produce nodules which progressively decrease in size as the interval between injection and examination increases. Eventually the dust disappears from the peritoneal tissue.

[1] From the Division of Industrial Hygiene, National Institute of Health.

[2] Director of the Bureau of Mines.

[3] Miller, J. W., and Sayers, R. R.: The response of peritoneal tissue to dusts introduced as foreign bodies. Pub. Health Rep., 49: 80–89 (January 19, 1934) (Reprint No. 1608). J. Am. Med. Assoc., 103: 907–912 (September 22, 1934). Am. J. Pub. Health, 25: 452–456 (April 1935). Pub. Health Rep., 51: 1677–1689 (December 4, 1936) (Reprint No. 1787).

Miller, J. W., and Sayers, R. R.: Microscopic appearance of experimentally produced dust nodules in the peritoneum. Pub. Health Rep., 50: 1619–1628 (November 15, 1935) (Reprint No. 1717).

Microscopically, a typical early nodule consists of a mass of the dust mixed with fine, granular, necrotic material. A zone of fibroblasts with an occasional macrophage surrounds this more or less centrally placed mass. With time the necrotic material becomes less and finally disappears. Brown pigment particles, apparently of endogenous origin, are usually found rather early, and in an experiment of a year's duration are the only evidence that the dust was introduced into the peritoneal cavity.

Dusts causing a proliferative type of reaction produce nodules which progressively increase in size as the interval between injection and examination increases. The maximum growth, using a 0.1-gm. dose for each guinea pig, is reached in about 90 days. Microscopically the nodules 7 days after injection are similar to those produced by the dusts of the absorptive group. As the process continues, the fibroblasts in the cellular zone about the central mass of dust and necrotic material are largely replaced by macrophages which are usually filled with dust particles. This is most marked in the 30-day series. Later, the engulfed dust particles appear to decrease in numbers and fibroblasts and adult connective tissue cells predominate. The area of necrotic material persists throughout the duration of the test. After 90 days fat cell formation in the cellular zone and calcification of the necrotic material is noted. All of the dusts classified in this group studied thus far are various forms of naturally occurring silica.

The nodules produced by the inert group of dusts are, in the early stages, grossly similar to those of the other two groups. As the interval between injection and examination increases the nodules become flattened with irregular edges, and numerous dispersed particles are present in the adjacent peritoneum. These are often found a considerable distance from the original nodules. The amount of dust found in the peritoneal cavity 1 year after injection is essentially the same as noted in 7 days. Histologically, the fibroblast is the early predominating cell. An increase in macrophages is noted at the 30-day interval and eventually fibrous tissue and accompanying fat cells predominate. No necrosis is noted at any interval in the entire process. The response is characteristic of that caused by a nonirritating foreign body.

It has been possible to correlate the response of peritoneal tissue to certain dusts with the results of X-ray examination or of post-mortem study of workers exposed by inhalation to high concentrations of the same dusts for protracted periods of time. These records are far from complete, because medical and roentgenographic surveys are available for only a limited number of the dusty trades. Nevertheless, the preliminary results of such comparisons can be summarized: (*a*) No cases of pneumoconiosis have been reported and confirmed among workers exposed solely to dusts of the absorptive group; (*b*) all

of the dusts so far examined that fall into the proliferative group are known to produce a nodular, pulmonary fibrosis (silicosis); (*c*) pneumoconioses caused by dusts of the inert group (asbestos,[4] anthracite mine dusts,[5] bisque ware,[6] mica,[7] pyrophyllite,[8] and talc [9]) have been reported as a result of X-ray examination of industrial workers. Where autopsy material is available, certain of the dusts of this group are known to produce a diffuse, interstitial, pulmonary fibrosis, or a mixed nodular and diffuse fibrosis, such as is produced by anthracite coal containing free silica.

Interpretation of the response produced by a dust in the peritoneal tissue in animals can be used as an index to determine the potential harmfulness of an industrial dust to which workers are exposed. Thus, an absorptive reaction can indicate that the dust is relatively harmless, while a proliferative response would indicate the dust to be definitely harmful. The dusts producing an inert reaction have been considered as less hazardous than those producing a proliferative reaction, and more dangerous than those of the absorptive group. The intraperitoneal method of studying the physiological action caused by dusts is not applicable to highly toxic material, a sublethal dose of which is too small to be grossly visible in the peritoneal tissue, or to dusts that are readily soluble.

The following dusts have been examined by this method and the results, with pertinent identifying data, are given below.

DUSTS CAUSING AN ABSORPTIVE REACTION

Calcite.—A pure mineral dust. Chemical analysis: Acid insoluble matter, 0.0 percent; silica, 0.0 percent. Petrographic examination: A calcite of high purity.

Calcite.—A pure mineral dust. Chemical analysis: Acid insoluble matter, 0.1 percent, all of which was silica. Petrographic examination: A calcite of high purity.

Precipitated calcium carbonate.—A chemical byproduct. An industrial dust. Chemical analysis: Silica, 0.4 percent; calcium carbonate, 87.9 percent; magnesium carbonate, 10.1 percent; magnesium oxide, 0.1 percent; iron and aluminum oxides, 0.6 percent. Petrographic examination: Precipitated calcium carbonate, about 98 percent; crystals, probably sodium carbonate, about 2 percent.

Gypsum.—The uncalcined, natural mineral. An industrial dust. Chemical analysis: Silica, 1.3 percent; calcium sulfate, 97.1 percent. Petrographic examination: Gypsum, about 70 percent; calcite, about 30 percent.

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4 Dreessen, W. C., DallaValle, J. M., et al.: A study of asbestosis in the asbestos textile industry. Pub. Health Bull. No. 241. U. S. Government Printing Office, 1938.

5 Sayers, R. R., Bloomfield, J. J., et al.: Anthracosilicosis among hard-coal miners. Pub. Health Bull. No. 221. U. S. Government Printing Office, 1936.

6 Flinn, R. H., Dreessen, W. C., et al.: Silicosis and lead poisoning among pottery workers. Pub. Health Bull. No. 244. U. S. Government Printing Office, 1939.

7 Dreessen, W. C., DallaValle, J. M., et al.: Pneumoconiosis among mica and pegmatite workers. Pub. Health Bull. No. 250. U. S. Government Printing Office, 1940.

8 Easom, H. F., Trice, M. F., and Carpenter, C. C.: A study of the effects of exposure to dust in the mining and milling of pyrophyllite. Report, North Carolina State Board of Health, February 1939.

9 Dreessen, W. C., and DallaValle, J. M.: Effects of exposure to dust in two Georgia talc mills and mines. Pub. Health Rep., 50: 131-143 (February 1, 1935) (Reprint No. 1669).

Limestone.—An industrial dust. Chemical analysis: Silica, 1.5 percent; calcium oxide, 54.4 percent; magnesium oxide, 0.4 percent; iron and aluminum oxides, 0.4 percent. Petrographic examination: Irregularly rounded calcite. No impurities noted.

Limestone.—An industrial dust. Chemical analysis: Silica, 2.73 percent; calcium carbonate, 95.21 percent; magnesium carbonate, 1.17 percent. Petrographic examination: A dolomitic limestone. No impurities observed.

Limestone.—An industrial dust. Chemical analysis: Acid insoluble matter, 7.2 percent; silica, 5 percent. Petrographic examination: Only an infrequent quartz crystal was noted. A high calcium carbonate content.

Limestone.—An industrial dust. Chemical analysis: Silica, 11.7 percent; calcium carbonate, 81.7 percent; magnesium oxide, 3.4 percent; ferric oxide, 1.4 percent; aluminum oxide, 1.5 percent. Petrographic examination: About 10 percent quartz and about 90 percent calcite.

Portland cement.—An industrial dust. Chemical analysis: Silica, 21.1 percent; calcium oxide, 74.4 percent; magnesium oxide, 2.8 percent. Petrographic examination: Normal portland cement.

Pyrolusite.—An industrial dust. Chemical analysis: Manganese, 54.9 percent. Petrographic examination: No quartz observed. This material was much more slowly absorbed than the others given here.

DUSTS CAUSING A PROLIFERATIVE REACTION

Bisque ware.—An industrial dust. Ground semivitreous pottery bisque ware, fired at a relatively low temperature. Chemical analysis: Silica, 72.0 percent. Petrographic examination: Quartz, about 40 to 50 percent. The remainder is semifused clay and feldspar.

Chert.—An industrial dust. Chemical analysis: Total silica, 76.1 percent. Petrographic examination: Quartz and chert, about 60 percent (about 25 percent of the silica is normal quartz). Calcite, about 40 percent.

Diatomite.—An industrial dust. Chemical analysis: Silica, 92.5 percent; aluminum oxide, 3.5 percent; ferric oxide, 1.5 percent; calcium oxide, 0.4 percent; magnesium oxide, 0.7 percent. Petrographic examination: Pure diatomite. No quartz or calcite present.

Greenware.—An industrial dust. Ground semivitreous, unfired pottery ware. Chemical analysis: Silica, 69.0 percent. Petrographic examination: Quartz, about 50 percent; feldspar, about 15 percent; clay, about 35 percent.

Greenware.—An industrial dust. Ground vitreous, unfired pottery ware. Petrographic examination: Higher quartz and less feldspar than the above. Clay, about the same amount.

Porcelain enamel frit.—An industrial dust. Chemical analysis: Silica, 35 to 50 percent; the remainder is oxides of antimony, zinc, and aluminum, and fluorides of sodium, aluminum, and calcium. Analysis varies within the above silica limits.

Quartz.—A pure mineral dust. Chemical analysis: Silica, 99.4 percent. Petrographic analysis: Normal crystalline quartz of high purity.

Quartz.—A pure mineral dust. Chemical analysis: Silica, 99.3 percent. Petrographic examination: Normal crystalline quartz of high purity.

Quartz.—An industrial dust. Chemical analysis: Silica, 99.1 percent. Petrographic examination: Normal quartz.

Quartz.—An industrial dust. Petrographic examination: Normal crystalline quartz of high purity.

Quartz.—An industrial dust. Identical with the above sample but treated with 0.6 percent crude pine fatty acids.

Quartz-sericite.—The source of this dust is not known. Chemical analysis: Total silica, 81.04 percent; calcium oxide, 0.30 percent; magnesium oxide, 0.45 percent; sodium oxide, 0.10 percent; potassium oxide, 0.98 percent; iron oxide, 0.25 percent; aluminum oxide, 14.26 percent; total water, 2.61 percent. Petrographic examination: Quartz, about 50 percent; muscovite (variety, sericite), about 45 percent; fibrous sericite, less than 5 percent.

Tripoli.—An industrial dust. Chemical analysis: Total silica, 98.9 percent; calcium oxide, 0.2 percent; magnesium oxide, 0.1 percent; iron and aluminum oxides, 0.3 percent. Petrographic examination: Chalcedonic silica (crystalline aggregates) with an occasional crystal of normal quartz.

DUSTS CAUSING AN INERT REACTION

Aluminum.—Pure aluminum bronzing powder of the finest grade. Chemical analysis: Aluminum oxide, 11.0 percent.

Alundum.—An industrial dust. Chemical analysis: Silica, 4.6 percent; aluminum oxide, 88.4 percent; ferric oxide, 6.9 percent. Petrographic examination: Well crystallized, artificial alumina.

Asbestos (amosite).—An industrial dust. Chemical analysis: Total silica, 48.31 percent; calcium oxide, 0.48 percent; magnesium oxide, 0.66 percent; sodium oxide, 0.72 percent; potassium oxide, 0.02 percent; iron oxide, 44.22 percent; combined oxides, 46.37 percent; total water, 3.62 percent. Petrographic examination showed predominating individual fibers and about 1 or 2 percent of dolomite.

Asbestos (chrysotile).—An industrial dust. Chemical analysis: Total silica, 37.52 percent; calcium oxide, 2.00 percent; magnesium oxide, 36.85 percent; sodium oxide, 0.54 percent; potassium oxide, 0.08 percent; iron oxide, 7.70 percent; combined oxides, 10.30 percent; total water, 12.86 percent. Petrographic examination: Serpentine, in part chrysotile, about 85 percent; dolomite, about 5 percent; magnetite and (or) chromite, about 5 percent; talc, less than 5 percent.

Asbestos (crocidolite).—An industrial dust. Chemical analysis: Total silica, 50.86 percent; calcium oxide, 0.68 percent; potassium oxide, 0.08 percent; iron oxide, 38.33 percent; combined oxides, 39.03 percent; total water, 5.02 percent. Petrographic examination showed fibrous material only.

Anthracite coal.—An industrial dust. Chemical analysis: Ash, 12.6 percent; silica, 6.6 percent. Petrographic examination: Coal about 95 percent; inorganic material, about 5 percent. About 60 percent of the inorganic material is quartz; about 40 percent is calcite, with an occasional crystal of rutile.

Anthracite coal.—An industrial dust. Chemical analysis: Ash, 16.0 percent; silica, 8.6 percent. Petrographic examination: Coal, about 95 percent; inorganic material, about 5 percent. About 95 percent of the inorganic material is quartz; about 5 percent is calcite, siderite, limonite, and rutile.

Bentonite.—An industrial dust. Petrographic examination: Clay, variety montmorillonite, about 97 percent; feldspar, about 2 percent; quartz, none observed.

Bisque ware.—An industrial dust. Ground vitreous pottery bisque ware, fired at a relatively high temperature. Petrographic examination: Quartz, about 30 to 40 percent. The particles are wholly or partially covered by the glass phase. This is absent in the semivitreous bisque ware.

Bituminous coal.—An industrial dust. Chemical analysis: Ash, 8.5 percent; silica, 0.8 percent. Petrographic examination: Mineral content (calcite), about 1 to 2 percent.

Bituminous coal.—An industrial dust. Chemical analysis: Ash, 8.0 percent; silica, 3.5 percent. Petrographic examination: Mineral content (quartz, calcite, clay), between 1 and 3 percent.

Calcium phosphate.—An industrial dust. Chemical analysis: Calcium phosphate, 75.38 percent; calcium carbonate, 3.98 percent; calcium fluoride, 6.80 percent; magnesium carbonate, 0.51 percent; iron oxide, 3.08 percent; aluminum oxide, 3.12 percent; free silica, 2.70 percent; combined silica, 1.87 percent. Petrographic examination: Earthy phosphates (not apatite), about 97 percent; normal and chalcedonic quartz, about 3 percent.

Chromite.—An industrial dust. Chemical analysis: Silica, 7.8 percent; chromic oxide, 25.0 percent. Petrographic examination: Quartz, less than 5 percent.

Diamond dust.—An industrial dust. Pure bortz diamond dust used as abrasive. Petrographic examination confirms identity.

Feldspar.—Chemical analysis: Total silica, 65.9 percent; calcium oxide, 0.81 percent; magnesium oxide, 0.10 percent; aluminum oxide, 19.55 percent; iron oxide, 0.28 percent; potassium oxide, 8.98 percent; sodium oxide, 3.18 percent. Petrographic examination: Feldspar (plagioclase-microcline), about 95 percent; normal quartz, about 5 percent.

Fuller's earth.—An industrial dust. Filtral clay. Chemical analysis: Silica, 55.7 percent; free silica (estimated), 1.0 percent; water, 15.9 percent. Petrographic examination: Clay and residual decomposing feldspar, about 95 percent; quartz, less than 1 percent; gypsum, less than 5 percent.

Fuller's earth.—An industrial dust. Chemical analysis: Silica, 56.4 percent; free silica (estimated), 7.0 percent; water, 8.5 percent. Petrographic examination: Clay and decomposing feldspar, about 90 to 95 percent; quartz, about 5 to 10 percent.

Fuller's earth.—An industrial dust. Chemical analysis: Silica, 57.9 percent; ferric oxide, 2.5 percent; aluminum oxide, 13.1 percent; calcium oxide, 2.9 percent; magnesium oxide, 8.5 percent; water, 6.7 percent. Petrographic examination: Clay-like masses, rounded and irregular, about 70 percent; quartz, about 15 percent; dolomite, about 15 percent.

Fuller's earth.—An industrial dust. Filtral clay. Chemical analysis: Silica, 62.1 percent; free silica (estimated), 3.0 percent; water, 14.9 percent. Petrographic examination: Clay and residual decomposing feldspar, about 98 percent; quartz, 1 to 2 percent; feldspar, an occasional fragment.

Glass wool.—An industrial dust. Finely ground sample of commercial hard glass wool was used. No chemical or petrographic examinations were thought necessary.

Hematite (jewelers' rouge).—An industrial dust. Chemical analysis: Total silica, 1.5 percent; iron oxide, 98.3 percent. Petrographic examination showed no impurities.

Kaolin.—An industrial dust. Petrographic examination: China clay and hydromica predominant; quartz and feldspar, a trace.

Lanthanum sublimate.—An industrial dust. From the burning of white flame electrodes. Chemical analysis: Lanthanum, 40.0 percent. Petrographic examination: Particles too small to identify.

Mica.—An industrial dust. Chemical analysis: Silica, 46.92 percent; magnesium oxide, 0.86 percent; aluminum oxide, 34.95 percent; ferric oxide, 2.65 percent; potassium oxide, 9.54 percent; sodium oxide, 1.02 percent; manganese dioxide, trace. Petrographic examination: Mica, both as plates and fibers, plates predominating, about 98 percent. A very small amount of quartz and feldspar.

Precipitator ash.—An industrial dust. Chemical analysis: Total silica, 49.86 percent; calcium oxide, 6.03 percent; magnesium oxide, 3.01 percent; iron and

aluminum oxides, 40.46 percent. Petrographic examination: Loosely consolidated, white, soft, grit-free ash, about 40 percent; partly rounded aggregates of semifused ash, about 45 percent; smooth fused glass globules, about 10 percent; normal quartz fragments, about 5 percent; unburned coal, less than 1 percent.

Precipitator ash.—An industrial dust. From the boiler plant of a coal company. Chemical analysis: Silica, 48.2 percent; aluminum oxide, 29.3 percent; ferric oxide, 8.5 percent; calcium oxide, 2.1 percent; magnesium oxide, 0.1 percent; organic matter, 8.6 percent. Petrographic examination: Predominantly spherulized glass, some coal fragments, and a trace of quartz.

Precipitator ash.—An industrial dust. Chemical analysis: Silica, 48.3 percent; aluminum oxide, 29.4 percent; ferric oxide, 8.6 percent; calcium oxide, 1.8 percent; magnesium oxide, 0.4 percent; organic matter, 8.7 percent. Petrographic examination: Predominantly semivitrified ash particles, some spheres, coal, and a trace of quartz.

Precipitator ash.—An industrial dust. Chemical analysis: Total silica, 44.7 percent; moisture, 0.1 percent. Petrographic examination: Mostly spherical fused-glass particles, with some semifused masses of crystallites, quartz, possibly calcite and coal fragments.

Precipitator ash.—An industrial dust. Lamphouse deposit from the burning of carbon arcs. Chemical analysis: Rare earth oxides (cerium group), 70.7 percent; ferric oxide, 0.8 percent; magnesium oxide, 0.5 percent; moisture, 9.8 percent; silica, none. Petrographic examination: Inorganic material, rudely rounded, about 5 percent of opaque carbonaceous material and no quartz.

Precipitator ash.—An industrial dust. Condensate from the flue system from burning of carbon arcs. Chemical analysis: Rare earth oxides (cerium group), 59.5 percent; silica, 1.0 percent; ferric oxide, 4.1 percent; magnesium oxide, 0.9 percent; calcium oxide, 0.6 percent; ignition loss, 18.4 percent. Petrographic examination: No quartz or calcite, otherwise similar to preceding sample.

Pyrophyllite.—An industrial dust. No chemical analysis obtained. Petrographic examination: Predominantly pyrophyllite, with a small amount of rutile and some quartz. The quantity of quartz was hard to estimate.

Rock wool.—An industrial dust. A finely ground sample of commercial, insulating rock wool.

Selenium.—An industrial dust. Chemical analysis: Selenium, 98.8 percent; tellurium, 0.01 percent; ash, 1.16 percent.

Selenium.—A chemically prepared sample of highest purity.

Sericite.—A pure mineral dust. Chemical analysis: Total silica, 51.74 percent; calcium oxide, 0.61 percent; magnesium oxide, 1.74 percent; sodium oxide, 3.40 percent; potassium oxide, 4.48 percent; iron oxide, 5.83 percent; combined oxides, 31.82 percent; total water, 6.26 percent. Petrographic examination: Sericite and feldspar residues (fibrous sericite predominates), about 95 percent; quartz, less than 5 percent.

Shale.—An industrial dust. Chemical analysis: Silica, 61.0 percent; aluminum oxide, 12.4 percent; calcium oxide, 4.5 percent; ferric oxide, 5.0 percent; magnesium oxide, 1.3 percent; sodium oxide, 2.3 percent; potassium oxide, 1.5 percent; moisture, 10.3 percent. Petrographic examination: About 35 percent quartz. The majority of the particles appear to be coated with clay.

Silicon carbide.—Pure manufactured silicon carbide. Chemical analysis: Silicon, 67.5 percent. Petrographic examination showed no impurities.

Soapstone.—An industrial dust. Chemical analysis: Total silica, 49.9 percent; calcium oxide, 1.7 percent; magnesium oxide, 26.2 percent. Petrographic examination: Talc, as plates or fibrous splinters, about 65 percent; tremolite, as long fibrous crystals, about 30 percent; dolomite, about 5 percent.

Soapstone.—An industrial dust. Chemical analysis: Total silica, 36.8 percent; calcium oxide, 5.0 percent; magnesium oxide, 22.7 percent. Petrographic examination: Talc, about 55 percent; dolomite, about 30 percent; tremolite, about 15 percent. No quartz observed.

Talc.—An industrial dust. Chemical analysis: Total silica, 49.0 percent; calcium oxide, 8.8 percent; magnesium oxide, 22.6 percent. Petrographic examination: Tremolite, about 60 percent; talc, about 40 percent.

Talc.—An industrial dust. Chemical analysis: Total silica, 56.54 percent; calcium oxide, 6.25 percent; magnesium oxide, 30.74 percent; calcium silicate, 11.00 percent; calcium carbonate, 1.88 percent; iron and aluminum oxides, 1.04 percent; ignition loss, 4.60 percent. Petrographic examination: Talc, mostly fibrous, about 75 percent; tremolite, partly altered to talc, about 25 percent; calcite and (or) dolomite, about 1 percent.

Titanium oxide.—An industrial dust. A finely divided high purity sample.

Sodium silicate.—A laboratory prepared sample containing 1 part sodium oxide to 3.1 parts silica. Higher ratios of sodium oxide kill the animals.

Trap rock.—An industrial dust. Chemical analysis: Silica, 51.7 percent; aluminum oxide, 16.0 percent; ferric oxide, 2.0 percent; ferrous oxide, 9.9 percent; calcium oxide, 10.0 percent; magnesium oxide, 6.2 percent. Petrographic examination: Feldspar, some slightly decomposed, about 45 percent; pyroxene, about 45 percent; magnetite, about 10 percent; biotite, about 1 percent.

Volcanic ash.—An industrial dust. Chemical analysis: Silica, 54.4 percent; aluminum oxide, 14.5 percent; ferric oxide, 3.8 percent; magnesium oxide, 2.6 percent; calcium oxide, 0.7 percent; ash, 78.2 percent. Petrographic examination: Fine volcanic ash partially altered to montmorillonite. No quartz observed.

Volcanic ash.—A specially treated sample. Chemical analysis: Silica, 74.3 percent; mixed oxides, 16.8 percent; ferric oxide, 2.2 percent; calcium oxide, 0.5 percent; magnesium oxide, 2.2 percent. Petrographic examination: Glass only. No quartz or calcite observed.

SUMMARY AND CONCLUSIONS

A definite quantity of dust injected into the peritoneal cavity of a guinea pig produces one of three types of reaction. It disappears, causes proliferation of the peritoneal tissue, or remains as an inert foreign body. These reactions may be used as a basis for the biological classification of industrial dusts, and seem to indicate that some relationship exists between the type of reaction produced in the peritoneal tissue by a dust and the ability of this dust to produce a characteristic type of pneumoconiosis. An absorptive reaction may indicate that a dust is relatively harmless, while a proliferative reaction, characteristic of quartz, may be associated with the ability to produce pulmonary fibrosis. Dusts of the inert group, that is, those that show a tendency to remain in the tissues, should be considered as potentially harmful, but not so dangerous as those causing a proliferative response.

With this biological method of classification, which in a number of instances has been correlated with clinical observations and industrial surveys, it is quite possible to determine the pneumoconiotic potentialities of a dust in a relatively short time, usually 60 days.

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IMMUNOLOGICAL RELATIONSHIPS BETWEEN THE RICKETTSIAE OF AUSTRALIAN AND AMERICAN "Q" FEVER

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INTRODUCTION

The relationship between Australian "Q" fever and a disease caused by an infectious agent isolated from ticks in Montana was first considered by Dyer (*1*) in a description of a human case of a disease probably contracted as a result of a laboratory infection by a member of the staff of the National Institute of Health while on a visit to the Rocky Mountain Laboratory of the United States Public Health Service at Hamilton, Mont. The source of the infection was problematical, although the subject had handled cultures and animals infected with a filter-passing agent which had been isolated at the laboratory by Davis and Cox (*2*) in 1935 from the wood tick *Dermacentor andersoni*. The organism concerned, as described by Cox (*3*), was a minute pleomorphic organism resembling the rickettsiae morphologically and in staining reactions, and in the intracellular and also extracellular occurrence of the organism in the affected tissues of laboratory animals. The infectious agent had been shown to be filterable through both Berkefeld N and W filters. In a later publication Cox (*4*) designated the new rickettsia as *Rickettsia diaporica*.

In experiments to determine the nature of the infectious agent it was found by Dyer at the National Institute of Health that cross-immunity tests between the virus from patient X which had been established in guinea pigs, and typhus, both epidemic and endemic, and Rocky Mountain spotted fever were negative, while five guinea pigs recovered from a strain of "Q" fever previously furnished to the National Institute of Health by Dr. Burnet of Australia were immune to the "X" strain of Dyer.

"Q" fever was described by Derrick (*5*) in 1937 as a new disease occurring in Australia. It affected principally meat workers and dairy farmers. It was distinguished from typhus by the absence of a rash and by a negative Weil-Felix reaction. The outstanding symptoms were fever and headache, and no fatalities occurred. Burnet and Freeman (*6*) described a rickettsial organism present in sections and smears of infected mouse spleens and livers. Emulsions of the organism were agglutinated by the serum of patients having the disease, and sera from convalescent patients protected laboratory animals against the disease. It was assigned the name *Rickettsia burneti* by Derrick (*7*).

Further cross-immunity and protection tests were later reported by Dyer (*8*). In the cross-immunity tests the strains used were: A "Q" fever strain received from Dr. Burnet in the form of two infected mouse spleens and subsequently maintained in mice and guinea pigs; the X strain of Dyer; a strain of endemic typhus and one of epidemic typhus, and two strains of Rocky Mountain spotted fever (the Bitterroot strain and an eastern spotted fever strain isolated from a case of the disease in Maryland). There was complete cross-immunity between the Q and X strains. There was no cross-protection between the X strain and the typhus and spotted fever strains, and none between the "Q" fever and spotted fever strains. There was a suggestion of immunity against the Q strain by the typhus strains, though the reverse was not true. In the protection tests definite protection against the X virus was shown with X serum and "Q" fever serum, while no protection was afforded against either by spotted fever serum.

Burnet and Freeman (*9*) also compared the Australian Q virus and the American X virus. They call attention to the more acute infection of guinea pigs by the American X strain, with shorter incubation period and death of the animals injected with the larger doses, the fibrinous exudate on the spleen, and congestion and partial consolidation of the lungs. Both were found virulent for monkeys, the X strain being found to be considerably more virulent. The higher virulence of the X strain was shown by the development of "foci" on the infected chorioallantoic membrane of chick embryos, whereas these were absent in embryos infected with the Q strain.

A complete cross-immunity in guinea pigs was obtained with the two strains; similar results were also obtained in agglutination tests with human, monkey, bandicoot, rabbit, and mouse sera against emulsions of rickettsiae from spleens of mice infected with both the Q and X strains.

EXPERIMENTAL

In an effort to elucidate further the relationship of the Australian and American diseases agglutination and agglutinin absorption tests were performed. Tests were also made with filtrates to determine whether a precipitin reaction could be demonstrated.

Two human sera were used, several rabbit immune sera against the X and Q strains, guinea pig and mouse sera, and also some specimens of sera received from Dr. Burnet of Australia, including sera from two convalescent patients and from two bandicoots.

The antigens were prepared principally from infected mouse spleens and livers, but yolk sac material of infected chick embryos was also used. The infection was established in mice by the intraperitoneal inoculation of 0.5 cc. of 10-percent suspensions of the spleen or liver of infected guinea pigs. Transfers were made at weekly intervals, using spleens or livers showing the largest number of rickettsiae. Usually two or three passages were necessary before the rickettsiae were present in sufficient numbers to warrant the preparation of the suspensions. It was found that the number of rickettsiae could be increased in a shorter length of time by the inoculation of mice with infected yolk sac material. In general, infection with a larger number of rickettsiae was established in a shorter time in the case of the X strain than with the Q strain; this was to be expected in view of the greater virulence of the X strain.

The infected mouse spleens and livers were ground in mortars with alundum, and 10-percent suspensions were prepared by the addition of buffered salt solution adjusted to pH 7.0. The method of Léon (*10*) was used in the separation of the tissues from the rickettsiae. After a preliminary centrifugation at 1,000 r. p. m. for 5 minutes to precipitate the alundum and larger particles of tissue the supernatant fluid was centrifuged at 3,500 r. p. m. for 1½ hours. The supernatant fluid from this centrifuging was retained for further centrifugation and for filtration. The majority of rickettsiae were precipitated by this method, but a few could be precipitated by added centrifugation at a high speed using the angle centrifuge at a speed of 10,000 r. p. m.

The precipitate was suspended in buffered salt solution at pH 7.0 and 0.5 percent glacial acetic acid was added, drop by drop, to a pH of 5.1 to 5.2 after the temperature had been brought to 35° to 40° C. A light centrifugation for 4 to 5 minutes served to precipitate the proteins, leaving the rickettsiae in the supernatant fluid with very little tissue. The suspensions were again centrifuged at 3,500 r. p. m. for 1½ hours to precipitate the rickettsiae and taken up in appropriate amounts of buffered salt solution and centrifuged lightly to precipitate any large particles. In some cases the reaction was adjusted with N/10 NaOH to pH 7.0 without recentrifugation.

Silica standards were used for adjusting the turbidity of the antigens and tests were carried out with suspensions with turbidities corresponding to 300 and 150 parts per million.

Suspensions were also prepared from infected yolk sac from chick embryos, employing the method described above, but more difficulty was experienced in obtaining pure suspensions with this material. As to the relative virulence of infected mouse spleen and infected yolk sac, the mouse spleen was found at times to be infective in a titer of 1×10^{-11}, which is the same as reported by Cox for yolk sac material.

For the immunizations of rabbits purified suspensions of the rickettsiae killed by the addition of 1/10,000 merthiolate were injected intravenously. Sera of rather good titer were obtained after two intravenous inoculations of 1 cc. of suspenions 2 days apart, followed by another inoculation of 2 cc. in a month, and bleeding in 2 weeks. Other rabbits were given a series of 6 inoculations at weekly intervals, without raising the titer. Another set of rabbits received inoculations with increasing amounts on 2 successive days each week for 8 weeks and in these somewhat higher titers were obtained in the case of the X serum but not of the Q serum (table 1).

AGGLUTINATION TESTS

Simple agglutination tests were performed with human sera and with sera of experimental animals as shown in table 1. The turbidity of the antigen was equivalent to 300 parts per million. Incubation was at 45° C. for 2 hours, after which the tubes were kept overnight at ice-box temperature.

TABLE 1.—*Agglutination of Q and X antigens by animal and human sera*

	Serum dilutions							Control (no serum)
	1:10	1:20	1:40	1:80	1:160	1:320	1:640	
X rabbit serum 1: [1]								
X antigen	4	4	4	4	4	4	2	0
Q antigen	3	3	4	4	4	4	2	0
X rabbit serum 5: [2]								
X antigen	4	4	4	4	4	4	3	
Q antigen	4	4	4	4	4	4	3	
Q rabbit serum 1: [1]								
X antigen	4	4	4	4	4	4	3	
Q antigen	4	4	4	4	4	4	3	
Q rabbit serum 4: [2]								
X antigen	4	4	4	4	4	3	2	
Q antigen	4	4	4	4	4	2	1	
X guinea pig serum 397: [3]								
X antigen	3	3	3	3	3	2	1	
Q antigen	3	3	3	3	3	2	1	
X guinea pig serum 413: [3]								
X antigen	3	3	3	3	3	2	1	
Q antigen	3	3	3	3	3	2	1	
Q guinea pig serum 388: [3]								
X antigen	4	4	4	4	4	3	1	
Q antigen	4	4	4	4	4	3	1	

See footnotes at end of table.

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TABLE 1.—*Agglutination of Q and X antigens by animal and human sera*—Contd.

	Serum dilutions							Control (no serum)
	1:10	1:20	1:40	1:80	1:160	1:320	1:640	
X mouse serum 18 (3):[4]								
X antigen	2	2	1	0	0	0	0	
Q antigen	2	1	1	0	0	0	0	
X mouse serum 18 (1-5):[4]								
X antigen	2	2	1	1	0	0	0	
Q antigen	2	2	2	1	0	0	0	
Human serum A:[5]								
X antigen	2	2	1	0	0	0	0	
Q antigen	2	2	1	0	0	0	0	
Human serum B:[5]								
X antigen	2	1	0	0	0	0	0	
Q antigen	2	1	0	0	0	0	0	
Human serum MacArthur:[6]								
X antigen	3	2	2	2	1	0	0	
Q antigen	2	2	2	1	1	0	0	
Bandicoot 119:[7]								
X antigen	2	1	0	0	0	0	0	
Q antigen	2	1	0	0	0	0	0	
Bandicoot 129:[7]								
X antigen	1	2	2	2	2	1	0	
Q antigen	2	2	2	2	1	0	0	
Normal rabbit serum:								
X antigen	0	0	0	0	0	0	0	
Q antigen	0	0	0	0	0	0	0	
Normal horse serum:								
X antigen	0	0	0	0	0	0	0	
Q antigen	0	0	0	0	0	0	0	

[1] Rabbits received 3 intravenous inoculations of rickettsia suspension.
[2] Rabbits received 16 intravenous inoculations of rickettsia suspension.
[3] Guinea pigs received 2 inoculations of 1 cc. of X vaccine a week apart and were tested for immunity 16 days later by inoculation of 1 cc. of a 10-percent suspension of infected guinea pig spleen.
[4] Mice inoculated intraperitoneally with 0.5 cc. of a 10-percent suspension of infected mouse spleen.
[5] Five months after onset of illness.
[6] Thirteen days after onset of illness (specimen received from Dr. Burnet).
[7] Specimen received from Dr. Burnet.

In another test an anti-X serum and an anti-Q serum were tested against two other lots of X and Q antigens. In this test the antigens were made up to a turbidity of 300 parts per million and to 150 parts per million with the result shown in table 2.

TABLE 2.—*Agglutination of Q and X antigens (of varying turbidity) by anti-Q and anti-X rabbit sera*

	Serum dilutions							Control (no serum)
	1:10	1:20	1:40	1:80	1:160	1:320	1:640	
X serum								
X antigen:								
300 p. p. m.	4	4	4	4	4	3	1	0
150 p. p. m.	4	4	4	4	4	4	1	0
Q antigen:								
300 p. p. m.	4	4	4	4	4	2	1	0
150 p. p. m.	4	4	4	4	4	2	1	0
Q serum								
X antigen:								
300 p. p. m.	4	4	4	3	2	1	0	
150 p. p. m.	4	4	4	4	2	1	0	
Q antigen:								
300 p. p. m.	4	4	4	4	2	1	0	
150 p. p. m.	4	4	4	4	3	1	0	

While the results obtained with the more dilute antigens were clear-cut it would not seem advisable to use as dilute a suspension as this in diagnostic tests with unknown sera.

The results of the simple agglutination test show the close relationship between the two rickettsiae, there being practically no difference in the results obtained, confirming the findings of Burnet and Freeman (*9*).

AGGLUTININ ABSORPTION TESTS

Agglutinin absorption tests were performed with absorbed X and Q serums against both X and Q antigens. The antigens were concentrated by subjecting 15 cc. of each suspension of a turbidity corresponding to 300 parts per million to high speed centrifugation for 30 minutes at approximately 10,000 r. p. m.; the supernatant fluid was removed and the precipitated rickettsiae suspended in 2 cc. of the 1:10 dilution of the corresponding serum. This suspension was placed in a 45° water bath for 2 hours and then centrifuged at a speed of 2,500 r. p. m. for 15 minutes to precipitate the agglutinated rickettsiae. The absorbed sera were tested against both the X and Q antigens of a turbidity of 300 parts per million with the results shown in table 3.

TABLE 3.—*Agglutinin absorption test*

	Serum dilutions						Control (no serum)
	1:20	1:40	1:80	1:160	1:320	1:640	
Absorbed X serum:							
X antigen	4	4	[illegible]	3	1	0	0
Q antigen	4	4	4	1	0	0	0
Absorbed Q serum:							
X antigen	1	0	0	0	0	0	
Q antigen	0	0	0	0	0	0	

The results against the X and Q antigens were similar with both absorbed sera. In the case of the Q serum the X and Q agglutinins were both absorbed and no agglutination was obtained against either antigen. With the X serum, however, it was necessary to repeat the absorption process twice, after which no agglutination was obtained against either antigen, as shown in table 4.

TABLE 4.—*Agglutinin absorption test with absorbed X serum*

	Serum dilutions					
	1:20	1:40	1:80	1:160	1:320	1:640
X antigen	0	0	0	0	0	0
Q antigen	0	0	0	0	0	0

The results of the agglutinin absorption tests are therefore further evidence of the identity of the two organisms.

TESTS FOR PRECIPITIN REACTIONS

Berkefeld N filtrates.—The supernatant fluids from the centrifugation of the 10 percent suspensions of mouse spleen and livers at 3,500 r. p. m. for 1½ hours were used for precipitin tests. These supernatant fluids were first filtered through Berkefeld N filters. It might be expected that such filtrates, while perfectly clear, would still contain a sufficient number of rickettsiae to cause agglutination. The results obtained are shown in table 5. In these tests an X serum with an agglutination titer of 1:640 and a Q serum with an agglutination titer of 1:320 were used. The concentrations of the serum and antigen were both varied in order to obtain information as to the most suitable dilution to use. A control test was made with normal rabbit serum.

As shown in the protocol of the test, a rather definite precipitate was formed, particularly in the lower dilutions. This was especially true of the X serum when tested against the Berkefeld N filtrates of the X and the Q supernatant fluids. The amount of the precipitate with the Q serum was decidedly less. Though the precipitate was definite and the supernatant fluid clear, the amount of precipitate formed was much smaller than in the agglutination test but somewhat greater in the case of the X serum than might perhaps be expected from residual rickettsiae in the filtrate.

A further test was made with the same filtrates passed through collodion membranes of a pore size of 0.4μ.[1] Burnet and Freeman (*6*) reported that the Q rickettsiae are not completely held back by gradocol membranes of 0.7μ average pore diameter but that small amounts passed through these membranes, as shown by inoculation and immunity tests on guinea pigs. Since the material used in the tests described had been treated with 1/10,000 dilution merthiolate it was necessary to test fresh material with the same pore-size filters to determine whether any of the infectious material passed through the filter. Suspensions of the fresh X material consisting of spleens and livers of infected mice were prepared as before and subjected to filtration through Berkefeld N filters before passing through the collodion membranes of the same pore size as was used for the filtrates under test. Infection occurred in guinea pigs with the Berkefeld filtrate as well as with the collodion filtered material, showing that some of the infectious agent was still present.

[1] The writer is indebted to Dr. Evelyn B. Tilden for the preparation of the collodion membranes.

Table 5.—*Tests for precipitin reactions (Berkefeld N filtrates)*

	Antigen dilutions							Control (no serum)
	1:2	1:4	1:8	1:16	1:32	1:64	1:128	
	X antigen							
X immune rabbit serum								
Serum dilutions:								
1:2	4	4	3	2	2	1	1	0
1:4	3	2	2	2	2	1	0	
1:8	2	2	1	1	1	0	0	
1:16	1	0	0	0	0	0	0	
1:32	0	0	0	0	0	0	0	
	Q antigen							
Serum dilutions:								
1:2	4	4	4	3	2	2	1	0
1:4	3	3	3	3	2	2	1	
1:8	3	3	2	2	1	0	0	
1:16	2	2	2	1	0	0	0	
1:32	2	1	1	0	0	0	0	
	X antigen							
Q immune rabbit serum								
Serum dilutions:								
1:2	2	2	1	1	0	0	0	
1:4	1	1	0	0	0	0	0	
1:8	0	0	0	0	0	0	0	
1:16	0	0	0	0	0	0	0	
1:32	0	0	0	0	0	0	0	
	Q antigen							
Serum dilutions:								
1:2	2	2	2	1	1	0	0	
1:4	1	1	1	0	0	0	0	
1:8	1	0	0	0	0	0	0	
1:16	0	0	0	0	0	0	0	
1:32	0	0	0	0	0	0	0	
	X antigen							
Normal rabbit serum								
Serum dilutions:								
1:2	1	0	0	0	0	0	0	
1:4	1	0	0	0	0	0	0	
1:8	0	0	0	0	0	0	0	
1:16	0	0	0	0	0	0	0	
1:32	0	0	0	0	0	0	0	
	Q antigen							
Serum dilutions:								
1:2	1	0	0	0	0	0	0	
1:4	0	0	0	0	0	0	0	
1:8	0	0	0	0	0	0	0	
1:16	0	0	0	0	0	0	0	
1:32	0	0	0	0	0	0	0	

However, the results obtained in the test with the immune sera were practically negative, as shown in table 6, indicating that under the conditions of the experiment precipitin was not present in the immune sera. It is possible that evidence of the presence of this antigen might be obtained by immunization of rabbits with Berkefeld filtrates of infected material. In any case, however, the results obtained with filtrates serve to establish further the identity of the two rickettsiae.

TABLE 6.—*Tests for precipitin reactions, X immune rabbit serum, and collodion membrane filtrates*

	Antigen dilutions			
	1:2	1:4	1:8	1:16
	X antigen			
Serum dilutions:				
1:2	2	1	0	0
1:4	1	0	0	0
1:8	1	0	0	0
1:16	0	0	0	0
	Q antigen			
Serum dilutions:				
1:2	2	1	0	0
1:4	1	0	0	0
1:8	1	0	0	0
1:16	0	0	0	0

DISCUSSION

The close immunological relationship of the Q and X strains of rickettsiae is indicated by the tests described. This affords added evidence of the identity of the Australian and the American diseases as shown by the cross-immunity and protection tests in laboratory animals described by Dyer (*1*, *8*) and by Burnet and Freeman (*9*).

As has been pointed out by Burnet and Freeman (*9*) and as has also been observed in this laboratory the virulence of the X strain is decidedly greater than that of the Q strain from Australia. This is reflected in the greater ease with which the disease may be established in mice, with correspondingly larger numbers of rickettsiae, as well as in the more pronounced effect in guinea pigs, with a high mortality where large doses of infected mouse spleens or livers are inoculated.

However, it is well known that in a number of other disease entities there may be a variation in the virulence of strains. This is particularly true of Rocky Mountain spotted fever, a much higher mortality in laboratory animals being associated with the Bitterroot type first described in the western part of the country than with certain other strains. Also it is well known that there may be fluctuations in the virulence of a particular disease at different periods; smallpox is a nota-

ble example. It is possible that the virulence of the Australian "Q" fever might differ from the similar disease in this country as a result of the modification of the virus in a host species or in an insect vector. The increased virulence of the X virus for the guinea pig after mouse passage, and the increased virulence for the mouse after chick embryo yolk sac passage afford concrete examples of such a change. In view, therefore, of the practical identity of the results in the serological tests, using both human and experimental animal sera, and of the results obtained in cross-immunity and cross-protection tests in animals, and of the clinical symptoms of the two diseases as pointed out by Dyer, it would appear justifiable to consider the Australian and the American types as one and the same.

SUMMARY

Agglutination and agglutinin absorption tests afford evidence of the identity of the rickettsiae which are the etiological agents of Australian "Q" fever, a disease affecting principally abattoir workers in that country, and a similar disease which occurred as the result of a probable laboratory infection in a member of the staff of the National Institute of Health. Further evidence of the identity of the two organisms has been shown in tests with immune and convalescent sera and Berkefeld N filtrates and ultrafiltrates, though this test was not shown to be that of a true precipitin reaction.

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THE INHIBITING EFFECT OF UREA ON THE MICROBIOLOGICAL ASSAY OF RIBOFLAVIN

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Fraser, Topping, and Isbell (*1*) applied the microbiological method of Snell and Strong (*2*) to the assay of riboflavin in the urine of normal and riboflavin-deficient dogs and rats. They found that the addition of increasing amounts of certain urines of low riboflavin content to the assay tubes produced a progressive diminution in the value of riboflavin found per milliliter of urine. A similar effect has been noted in the assay of human urines of low riboflavin content. Three typical examples of the inhibiting action of human urine are presented in table 1.

TABLE 1.—*Inhibitory effect of urine on the microbiological assay of riboflavin*

Ml. urine added to tube	Micrograms riboflavin found by assay					
	I		II		III	
	Per tube	Per ml.	Per tube	Per ml.	Per tube	Per ml.
1	0.07	0.07	0.075	0.075	0.06	0.06
2	.136	.068	.14	0.07	.08	.04
3	.183	.061	.15	0.05	.09	.03
4	.2	.05	.16	0.04	.1	.02
5	.15	.03	.15	0.03	----------	----------

In an effort to determine the cause or causes of the inhibitory effect of urine on the microbiological assay certain quantitatively important constituents of urine were studied for their inhibitory action on acid production by *Lactobacillus casei*. Definite quantities of the pure compounds were added to tubes containing known amounts of riboflavin. The quantities added were chosen to cover and exceed the range over which the ions comprising the compounds, or the compounds themselves, occur in human urine. NaCl in amounts from 10–250 milligrams, Na_2SO_4 in amounts from 10 to 200 milligrams, KCl in amounts from 10 to 100 milligrams $(NH_4)_2SO_4$ and NH_4Cl in amounts from 10 to 200 milligrams were tested. No diminution in the assay values was noted with any of these salts over the ranges used. Some increase in the assay values was found with all the salts at levels of 80 milligrams or more per tube.

Addition of increasing amounts of urea to the tubes produced a progressive decrease in the assay values from approximately 20 percent at the level of 20 milligrams of urea per tube to 80 to 100 percent at the level of 80 milligrams. Table 2 shows the mean values of riboflavin found by assay in the presence of varying amounts of urea.

TABLE 2.—*Effect of urea on microbiological assay of riboflavin* [1]

Micrograms riboflavin added to tube	Micrograms riboflavin found in presence of urea					
	Milligrams urea added to tube					
	10	20	30	40	60	80
0.05	0.045	0.04	0.04	0.035	0.02	0.00
.075	.06	.05	.04	.04	.02	.00
.1	.09	.08	.07	.06	.05	.04
.15	.14	.13	.11	.09	.08	.055
.2	.19	.165	.15	.135	.12	----------
.25	.2	.2	.195	.16	----------	----------
.3	.25	.24	.21	.175	----------	----------

[1] Each value is an average of 5 to 8 duplicate determinations.

From the data given in table 2 the partial regression equation (*3*),

$$X = 0.000824Y + 1.21Z - \pm 0.017,^{1}$$

was calculated where X represents the micrograms of riboflavin actually present in the tube, Y the milligrams of urea present, and Z the amount of riboflavin apparently present as determined by assay.

Since the equation was derived from data based on the depressing action of a pure solution of urea, it was necessary to determine whether or not urea accounted for all the inhibiting effect of urine. Specimens of urine exhibiting the inhibitory phenomenon were therefore assayed, the amount of urea per milliliter of urine determined by the method of Van Slyke and Cope (*4*),[2] and the values obtained corrected by the use of the regression equation. Known amounts of riboflavin were added to the same urines, assays performed, the values corrected by use of the regression equation, and the percentage recovery of added riboflavin calculated. Table 3 gives the results obtained with 5 typical urines at varied levels of both urea and riboflavin.

One hundred and thirty-six determinations on 24 separate urines gave an average recovery of added riboflavin of 103 percent with a variation of 87 to 118 percent.

In 48 other determinations on 8 urines, known amounts of riboflavin were added to tubes containing 0.5 to 1.0 milliliter of urine. The urines used contained less than 20 milligrams of urea per milliliter of urine. The tubes were assayed, the value of riboflavin per milliliter of urine obtained by difference, and the results obtained compared with those found by using the correction formula. The average values found were identical in all cases.

[1] Standard error of estimate.

[2] The method was slightly modified from the procedure described by Van Slyke and Cope in that the urine was diluted 1–50 or 1–100 instead of to a value calculated from the per minute urine volume and read against the standard of 0.2 milligrams nitrogen, instead of against a blood filtrate.

TABLE 3.—*Recoveries of riboflavin added to urine*

Urine No.	Ml. urine added	Mg. urea added by urine	Micrograms riboflavin added by urine	Micrograms riboflavin added as pure solution	Total riboflavin present in tube	Micrograms riboflavin actually found	Micrograms riboflavin by correction formula	Percent recovery
IV	2.0	15.8	0.06	0.05	0.11	0.09	0.1	90
	2.0	15.8	.06	.1	.16	.15	.16	100
	3.0	23.7	.09	.05	.14	.12	.14	100
	3.0	23.7	.09	.1	.19	.16	.18	95
	4.0	31.6	.12	.05	.17	.13	.15	90
V	2.0	22.4	.04	.1	.14	.13	.14	100
	3.0	34.1	.06	.15	.21	.17	.21	100
	4.0	42.8	.08	.2	.28	.19	.24	87
VI	3.0	36.3	.045	.1	.145	.13	.16	110
	3.0	36.3	.045	.15	.195	.17	.2	98
	3.0	36.3	.045	.2	.245	.208	.235	96
	4.0	48.4	.06	.1	.16	.153	.19	118
VII	3.0	46.5	.135	.05	.185	.157	.195	105
	4.0	71.5	.18	.05	.23	.171	.24	104
VIII	1.0	23	.07	.05	.12	.12	.14	116
	2.0	46	.14	.1	.24	.21	.25	104

DISCUSSION

The excellent recoveries of added riboflavin from urines exhibiting the depressing effect are strong evidence that urea accounts for most, if not all, of the inhibiting action of urine. The results given also prove that the regression equation may be used to obtain the true amount of riboflavin present in a urine exhibiting the depressing phenomenon. The equation need not be applied unless 20 milligrams or more of urea are present in each tube. The regression equation applies best between the levels of 20 to 60 milligrams of urea and in the presence of 0.075 to 0.2 micrograms of riboflavin. If desired, the use of the regression equation may be avoided altogether by adding known quantities of riboflavin to tubes containing 0.5 to 1.0 milliliter of urine and obtaining the values per milliliter of urine by difference.

The stimulation observed with various inorganic salts at levels of 80 milligrams or more should not introduce appreciable error since the amounts required to produce the stimulation do not ordinarily occur in human urine (*5*).

SUMMARY

The inhibiting effect of urines of low riboflavin content on the microbiological assay for riboflavin according to the method of Snell and Strong is demonstrated. Methods for correcting the error due to the inhibiting effect of urea are presented.

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STUDIES ON THE NATURAL HISTORY OF THE VIRUS OF LYMPHOCYTIC CHORIOMENINGITIS IN MICE

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The virus which produces lymphocytic choriomeningitis in man occurs spontaneously in domestic mice (*Mus musculus*), and this rodent infection is connected epidemiologically with human cases (Armstrong). The studies here reported deal with the behavior of the infection in mice.

Spontaneous infection in white mice was studied intensively by Traub, who found that less than 20 percent of the naturally infected animals showed symptoms, although virus was present in practically every organ of the infected mice, as well as in blood, urine, and nasal secretions. Infection spread among mice in two ways—transmission from mother to offspring *in utero*, and from infected to noninfected mice by contact. Mice infected *in utero* or in extreme infancy often retained virus for long periods, but when mice were infected after reaching maturity, virus was recoverable for only a short period. Exposure to the virus produced strong immunity in mice, regardless of whether the animal in question continued to harbor demonstrable virus or not.

The present report is essentially a confirmation of Traub's thorough studies, with some extensions consequent to a somewhat different approach.

METHOD OF STUDY

Except where otherwise mentioned, these studies were made on albino mice of the National Institute of Health "Swiss" stock. Usually mice were kept in glass battery jars; where more than 6 mice were used at a time, large glass cages with screen tops were employed.

Two strains of virus were used, one recently isolated from a human case of choriomeningitis, and the other originating in naturally infected house mice. The two strains behaved similarly.

To determine whether mice under study had contracted infection, the usual method was to test their ability to withstand intracerebral

inoculation with 10–15 M. L. D. of the stock virus; along with each group of mice thus tested, from 5 to 15 fresh mice were inoculated in the same manner, in order to make certain that the virus used in the immunity test actually produced the disease in animals known to be nonimmune. In some cases, mice were sacrificed and active virus was recovered from their viscera.

In this report the term "natural infection" refers to infection contracted *in utero* or by contact with infected mice, as distinguished from infection by inoculation.

NATURE OF THE INFECTION IN MICE

Symptoms in naturally infected mice were extremely mild or entirely inapparent, as shown by the following examples:

1. Seven naturally infected house mice kept in the laboratory for over 5 months showed no evidence of illness, though virus was recovered from their blood and feces during this period. Thirty-six white mice infected by cage contact with the 7 house mice also showed no symptoms.

2. In a series of experiments, 66 fresh white mice were placed in jars with infected white mice for periods of 12 to 28 days; over half became infected through this contact, and there were 4 deaths, presumably incidental, since in another series of tests, where no transmission of infection occurred in mice kept under the same conditions, there were 24 deaths among 117 animals.

Mice from infected litters seemed to mature less rapidly than did normal mice, though this is only an impression, as no weights were kept.

TRANSMISSION AND SURVIVAL OF INFECTION

Transmission in utero.—Infection of mice *in utero* was accomplished in two ways: (1) Pregnant mice inoculated before delivery produced infected litters in many instances; the route of inoculation of the mother was not important. (2) Mice were inoculated intranasally 1 or 2 days after birth; when the females matured and were bred, they tended to produce infected offspring.

Mice infected *in utero* transmitted the virus to their descendants in many instances, as is shown in table 1. That infection of these offspring was not due to chance spread of virus in the laboratory appears later, in table 3, where it is shown that litters born to mice inoculated after reaching adulthood, but before becoming pregnant, failed to become infected.

Since infected mice were usually detected by immunity tests, it is necessary to show that it was actual infection and not merely immunity that was passed on from mothers to offspring. This is indicated by the following observations:

1. One mouse from each of 4 litters, removed two or three generations from inoculated ancestors, was killed and found to contain virus by inoculating an emulsion of its spleen and liver into fresh mice; the litter mates of these infected mice were at the same time found to be immune by the usual test. On the other hand, one mouse from each of two other litters yielded no virus, and the litter mates of these mice did not survive the immunity test.

2. Five mice infected *in utero* were killed from 107 to 216 days after birth and found still to harbor living virus. [1]

3. Virus was recovered from a pooled sample of feces from 5 mice infected *in utero;* the mice were 107 days old at the time of this test.

4. Twenty-one mice infected *in utero* transmitted infection to fresh mice kept in jars with them for periods of 12 to 28 days.

TABLE 1.—*Transmission of virus to descendants of mice inoculated during pregnancy or during infancy*

Descendants of inoculated female mice	24 mice inoculated 1 to 11 days before delivering young				21 mice inoculated intranasally within 1 to 2 days of birth			
	Total born		Number infected		Total born		Number infected	
	Litters	Mice	Litters	Mice	Litters	Mice	Litters	Mice
First generation	14	64	10	34	17	65	15	57
Second generation	23	116	10	56	9	50	9	50
Third generation	14	69	12	57	(1)	(1)	(1)	(1)

1 Studies on the group infected by inoculation in infancy were not carried beyond two generations of offspring.

The prolonged survival of the virus in mice infected *in utero* is indicated by these observations. Another example of this survival appears in the ability of females to transmit infection to successive litters in the same generation. This is shown in table 2.

TABLE 2.—*Transmission of infection by mice to multiple litters*

Offspring of 8 mice infected *in utero* or early infancy, which gave birth to more than 1 litter each	Number mice in litters	Number mice infected
First litters	32	32
Second litters	46	[1] 33
Third litter	2	2

1 Offspring of 6 mice. Two mice produced infected first litters but failed to infect their second litters.

In contrast to mice infected *in utero* or early infancy, animals inoculated after reaching maturity (i. e., 3 weeks or older) did not transmit infection to their offspring, provided they were not pregnant at the time of inoculation or did not become so shortly thereafter. This appears in table 3.

[1] These mice had been inoculated with the stock virus when they were 1 month old, in order to test their immunity. Experience with this strain of virus has indicated that in the majority of instances it is not recoverable from inoculated mice for longer than a few weeks after injection, and therefore could not have been responsible for the results obtained here.

TABLE 3.—*Failure of mice inoculated after reaching maturity to transmit infection to their offspring*

Mice inoculated during adult life and subsequently producing offspring	Offspring			
	Number born		Number infected	
	Litters	Mice	Litters	Mice
33 females inoculated 34 to 149 days before giving birth to young.	26	142	None	None
14 males inoculated 28 to 95 days before siring offspring	8	39	None	None

One female, not included in table 3, produced infected offspring 31 days after being inoculated; apparently enough virus survived to infect the offspring conceived shortly after inoculation.

In further contrast to mice infected *in utero* or early infancy, adult mice inoculated while pregnant did not transmit the virus to offspring conceived after the birth of those carried at the time of inoculation. Three mice inoculated during pregnancy and giving birth to infected litters subsequently produced additional offspring to the total of 16, none of which were infected.

Most of the second and third generations of infected mice were obtained by breeding infected males with infected females. In three instances, however, naturally infected females produced litters sired by uninfected males. Two of these litters were infected, indicating that virus passed from infected mothers to offspring regardless of the status of the male parents. On the other hand, when infected males were bred to uninfected females, the offspring were not infected, as indicated by the following summary: 11 infected males were bred with 19 uninfected females; 13 litters resulted, comprising 74 mice; 12 of these litters, comprising 69 mice, were uninfected. One litter of 5 mice was immune when tested, and it is probable that these particular mice acquired infection through contact with the infected father, a circumstance generally prevented by removing the male before the young were delivered. A second uninfected female in the same jar at the time this litter was born later produced an uninfected litter, contact between this litter and the father having been avoided.

Transmission by contact.—Mice infected *in utero* or in infancy transmitted infection to others placed in contact with them. This is shown in table 4.

In one of the tests in which white mice were infected through contact with the gray mice, virus was recovered from a contact as early as the sixth day.

In addition to the examples of contact infection given above, there were four litters inoculated intranasally on the first or second day after birth and allowed to remain with the mothers for a month. At the end of this period all the mothers were immune.

TABLE 4.—*Transmission of virus by mice infected in utero or infancy to mice placed in contact with them*

Infected mice with which fresh mice were in contact	Length of contact (days)	Fresh mice	
		Number used	Number infected
7 gray house mice infected in nature	6 to 28	[1] 47	36
32 white mice infected *in utero* and early infancy [3]	12 to 28	[2] 66	35

[1] 6 different tests.
[2] 18 different tests.
[3] These mice were all at least 1 month old when used for these tests.

Mice which became infected after reaching their maturity (i. e., 3 weeks or older) rarely transmitted the virus to contacts, in contrast to mice infected *in utero* or in infancy. This was true regardless of whether the adult mice had been infected by inoculation or by having been themselves in contact with mice capable of transmitting infection. The experiments establishing these statements are summarized in table 5.

TABLE 5.—*Failure of mice infected after reaching maturity (i. e., 3 weeks or older) to transmit virus to fresh mice placed in contact with them*

Mice infected by inoculation or by contact after reaching maturity, and then placed in contact with fresh mice	Length of contact (days)	Fresh mice	
		Number used	Number infected
25 white mice infected through contact with naturally infected mice	16 to 31	[1] 50	2
72 white mice infected by inoculation (various routes)	8 to 37	[2] 95	1
Total		145	3

[1] 10 different tests.
[2] 23 different tests.

The mode of spread of contact infection among mice was investigated by the following experiments:

1. *Sex.*—Semen taken from infected males and instilled into the vaginae of 12 females infected 9 of them; the females eventually produced 8 litters, comprising 51 mice, none of which were infected. Experiments on contact infection indicated that sexual contact was not necessary for transmission of the virus: 6 females infected 8 out of 17 males; 5 males infected 9 out of 16 females; 2 males infected 3 out of 5 males; 4 females infected 9 out of 16 females.

2. *Feces and urine.*—Traub found the virus in urine of infected mice but was unable to infect mice by placing them in cages heavily contaminated with such urine. During the present studies, virus was recovered from two pooled samples of feces collected, respectively, from 3 and 5 infected mice. The experiments summarized below,

however, indicate that feces and urine were not essential for transmission of the infection among mice:

(*a*) Ten fresh mice were put into a glass cage which had been inhabited for 22 days by infected mice, and which had not been cleaned in any way. After 16 days in this cage the fresh mice were found to be nonimmune; the infected mice had transmitted infection to 18 fresh mice during their occupancy of this cage.

(*b*) Each of five jars was divided into an upper and a lower compartment by a horizontal screen. In each of three jars an infected mouse was kept in the bottom compartment and two fresh mice in the top; in the other two jars the position of the mice was reversed. Mice in the lower compartment were exposed to urine and feces falling through the screen from those above, but mice in the upper section were not exposed to these excreta. The test lasted a month, and the result was that one fresh mouse in each jar was found to have become infected. In other words, mice not exposed to feces and urine were infected as readily as those so exposed.

It must be concluded from these experiments that neither sexual contact nor feces and urine were essential in the spread of contact infection among mice. Since ectoparasites were not present in the cages where such spread occurred, it appears that nasal discharges or saliva were the likely means of disseminating the virus. Quite possibly both are important; Traub found abundant virus in nasal washings, and nose-to-nose contact among mice is common. In some instances, however, fighting results in infliction of numerous wounds, and during these studies virus was recovered from 2 badly bitten mice.

Survival of the virus.—The observations already discussed suggest that virus survived for long periods in mice infected *in utero* or in early infancy, whereas in mice infected after reaching maturity active infection tended to be demonstrable only for a short while. This contrast between mice infected at different stages of life is further emphasized by the following experiment:

Five fresh mice were placed in a cage with 3 infected mice, 1 of which had been infected *in utero* and 2 on the day of birth. After 23 days in the cage, all the mice were given the usual immunity test, which they survived; 35 days after this test all were killed and tested for virus. The mouse infected *in utero*, which was 216 days old when killed, yielded active virus, as did one of those infected on the day of birth, 185 days before; the other mouse infected on the day of birth, 148 days previously, yielded no demonstrably active virus, nor did any of the 5 mice which had developed an immunity following contact with the infected animals.[2]

[2] Mice inoculated intracerebrally with spleen emulsions of these five mice failed to develop any signs of choriomeningitis; the mice inoculated with such an emulsionf rom one of these five mice later survived an immunity test with the stock virus, indicating that in this one instance virus may have been present in very small amount or in a condition not sufficiently active to produce detectable disease.

It appears that there was a basic difference between the nature of infection established *in utero* or in very young mice and that introduced into mice already mature, and this difference was indicated by the ability of mice infected *in utero* or in infancy to retain the virus and to transmit infection to offspring and contacts, while the others did not.

SUMMARY

Infection of white mice by the virus of choriomeningitis, when acquired *in utero* or by contact, was generally of an inapparent type.

Mice infected *in utero* or early infancy tended to retain active virus for long periods, probably in some instances for life, and to transmit infection to offspring and contacts. Infection passed from infected mothers to offspring whether the fathers were infected or not, but it did not pass from infected fathers to offspring through uninfected mothers.

Mice infected after reaching maturity did not transmit infection to their offspring, except for females pregnant at time of inoculation, and rarely infected contacts. Active virus was not generally demonstrable in such mice except for short periods after exposure or inoculation.

Transmission of infection from naturally infected mice to fresh contacts occurred when exposure through sexual contact, urine, and feces were eliminated, infection in such instances apparently being conveyed by nasal secretions or saliva.

These observations are in agreement, in the essential points, with those previously reported by Traub.

The behavior of this virus in mice is particularly interesting because of two underlying facts: First, the continuous propagation of an infection that is inapparent, or nearly so; and second, the basic difference in response to infection shown by very young mice as compared to the response of mice subjected to infection after reaching maturity.

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A NOTE ON MODIFIED RADIO PRATIQUE IN GUAYAQUIL

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Radio pratique was inaugurated at the port of New York on February 1, 1937, and has been in successful operation since that time.[1] This procedure is also practiced in Boston, New Orleans, New York, Los Angeles, and San Francisco.

In United States ports only passenger vessels with accredited ship's doctors are eligible for radio pratique. However, in Canada cargo as well as passenger vessels are accorded this privilege even though a physician is not a member of the crew. Radio pratique, as administered at William Head quarantine station, Victoria, British Columbia, has been described by Dr. H. B. Jeffs.[2] Experience with radio pratique for freighters has been entirely satisfactory in Canada.

Radio pratique principles have now been extended to other countries, Ecuador being the latest to make use of this practical modification of maritime quarantine. According to recent information the following procedure is in effect for southbound vessels arriving in Guayaquil:

1. Within 24 hours prior to arrival the ship's doctor shall send a radio message to the quarantine officer advising that there is no illness of any kind on board and that all passengers have been vaccinated against smallpox.

2. Upon arrival in port the vessel will be boarded by the quarantine officer who will require a copy of the radiogram signed by the master and the ship's doctor.

3. The ship's doctor must also present a vaccination certificate for each person who lands in Guayaquil.

4. Having obtained these documents the quarantine officer may allow the other port authorities to board the vessel without further formality.

5. Pratique will be withheld and customary inspection made under the following circumstances:

[1] Akin, C. V.: Pratique by Wireless in Lieu of Quarantine Inspection for Passenger Vessels. Pub. Health Rep., 52: 507 (Apr. 23, 1937).
[2] Bulletin of the International Office of Public Health, 31: 1581 (September 1939).

(*a*) If the radiogram is not confirmed.

(*b*) If the vaccination certificates are not in order.

(*c*) If illness has occurred after the radiogram was sent.

Cargo vessels are not included in this procedure.

Steamship companies estimate that a considerable saving of time will be effected at Guayaquil by this utilization of radio messages prior to the arrival of vessels in port.

Comment.—It has been found, as the result of actual practice and careful observation, that the public health has not been imperiled by radio pratique and that this modification of maritime quarantine is helpful in expediting commercial activities. With the leadership already provided in several countries it may be expected that the measure will be adopted even more widely.

NOTIFIABLE DISEASES IN THE UNITED STATES, 1939

Morbidity and Mortality Summaries for Certain Important Communicable Diseases

The United States Public Health Service has recently issued a tabular morbidity and mortality compilation, by States and by months, for the notifiable diseases as reported by the State health officers in 1939.[1] A summary of this compilation is presented here, together with case and death rates, case fatality rates, and, in some instances, the estimated expectancy based on figures for recent preceding years.

For certain diseases, some States do not report cases, or the case reports are manifestly incomplete. In such instances groups of States with the most satisfactory morbidity reports are treated separately in order to arrive at more nearly accurate case and case fatality rates, while the totals for the larger group of States include the deaths as cases in States which reported fewer cases than deaths. Case fatality rates are not computed, however, on such totals.

The mortality figures may be considered as approximately correct, although they will not agree in all instances with the final figures of the Bureau of the Census.

The estimated expectancy, given for some of the diseases, represents an attempt to ascertain from the experience of recent years the number of cases of a disease that might normally have been expected in 1939.

In comparing the numbers of cases reported in 1939 with the estimated expectancy, or with figures for preceding years, it should be borne in mind that there has been a gradual improvement in the

[1] The Notifiable Diseases—Prevalence in States, 1939. Supplement No. 163 to the Public Health Reports. Government Printing Office, Washington, 1941.

reporting of notifiable diseases and that the population has increased. A large increase, however, especially in the case rate, is likely to represent an actual increase in the prevalence of the disease. The populations given for groups of States, used in computing case and death rates, were estimated as of July 1, 1939, by the Public Health Service, and are based on the populations for 1930 and preliminary figures for 1940 populations as published by the Bureau of the Census.

CHICKENPOX (38e) *

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	258,486
Estimated expectancy based on years 1932-38	261,519
Cases per 1,000 inhabitants, 1939	1.984
Cases per 1,000 inhabitants, estimated expectancy	2.059
Deaths registered, 1939	110
Deaths per 1,000 inhabitants, 1939	0.001
Cases reported for each death registered, 1939	2,350
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	258,746
Cases per 1,000 inhabitants, 1939	1.979

DIPHTHERIA (10)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	24,045
Estimated expectancy based on years 1932-38	38,269
Cases per 1,000 inhabitants, 1939	0.185
Cases per 1,000 inhabitants, estimated expectancy	0.301
Deaths registered, 1939	2,022
Deaths per 1,000 inhabitants, 1939	0.016
Cases reported for each death registered, 1939	12
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	24,053
Cases per 1,000 inhabitants, 1939	0.184

DYSENTERY (AMOEBIC) (27b)

33 States:[1]	
Cases reported, 1939 (population 107,355,000)	2,981
Cases per 1,000 inhabitants, 1939	0.028
Deaths registered, 1939	220
Deaths per 1,000 inhabitants, 1939	0.002
Cases reported for each death registered, 1939	14
39 States:[1]	
Cases reported, 1939 (population 126,553,000)	[2] 3,039
Deaths registered, 1939	278
Deaths per 1,000 inhabitants, 1939	0.002
47 States:[1]	
Deaths registered, 1939 (population 130,275,000)	282
Deaths per 1,000 inhabitants, 1939	0.002

DYSENTERY (BACILLARY) (27a)

31 States:	
Cases reported, 1939 (population 101,476,000)	21,137
Cases per 1,000 inhabitants, 1939	0.208
Deaths registered, 1939	831
Deaths per 1,000 inhabitants, 1939	0.008
Cases reported for each death registered, 1939	25
41 States:[1]	
Cases reported, 1939 (population 126,719,000)	[2] 21,327
Deaths registered, 1939	1,021
Deaths per 1,000 inhabitants, 1939	0.008
46 States:[1]	
Deaths registered, 1939 (population 128,382,000)	1,046
Deaths per 1,000 inhabitants, 1939	0.008

ENCEPHALITIS, EPIDEMIC OR LETHARGIC (37)

29 States:[1]	
Cases reported, 1939 (population 81,496,000)	787
Cases per 1,000 inhabitants, 1939	0.010
Deaths registered, 1939	363
Deaths per 1,000 inhabitants, 1939	0.004
Cases reported for each death registered, 1939	2.168
47 States:[1]	
Cases reported, 1939 (population 130,275,000)	[2] 1,069
Deaths registered, 1939	645
Deaths per 1,000 inhabitants, 1939	0.005

*Figures in parentheses in the subheadings are disease title numbers from the International List of Causes of Death, 1938.

[1] The District of Columbia is also included but not counted as a State.

[2] Includes the number of deaths used as cases when no cases are reported, or when the reported number of cases is less than the number of deaths.

GONORRHEA (25)

48 States:[1]	
Cases reported, 1939 (population 130,763,000)	178, 343
Cases per 1,000 inhabitants, 1939	1. 364

INFLUENZA (33)

42 States:[1]	
Cases reported, 1939 (population 101,802,000)	275, 503
Cases per 1,000 inhabitants, 1939	2. 706
Deaths registered, 1939	19, 724
Deaths per 1,000 inhabitants, 1939	0. 194
Cases reported for each death registered, 1939	13. 968
47 States:[1]	
Cases reported, 1939 (population 130,275,000)	[2] 277, 613
Deaths registered, 1939	21, 834
Deaths per 1,000 inhabitants, 1939	0. 168
48 States:[1]	
Cases reported, 1939	[2] 277,616

MALARIA (28)

40 States:	
Cases reported, 1939 (population 125,977,000)	82, 654
Cases per 1,000 inhabitants, 1939	0. 656
Deaths registered, 1939	1, 749
Deaths per 1,000 inhabitants, 1939	0. 014
Cases reported for each death registered, 1939	47
40 States:[1]	
Cases reported, 1939 (population 126,627,000)	[2] 82, 655
Deaths registered, 1939	1, 750
Deaths per 1,000 inhabitants, 1939	0. 014
47 States:[1]	
Deaths registered, 1939 (population 130,275,000)	1, 750
Deaths per 1,000 inhabitants, 1939	0. 013

MEASLES (35)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	403, 037
Cases per 1,000 inhabitants, 1939	3. 094
Deaths registered, 1939	1, 171
Deaths per 1,000 inhabitants, 1939	0. 009
Cases reported for each death registered, 1939	344
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	403, 317
Cases per 1,000 inhabitants, 1939	3. 084

MENINGITIS, MENINGOCOCCUS (6)

45 States:[1]	
Cases reported, 1939 (population 128,024,000)	1, 970
Estimated expectancy based on years 1932-38	3, 611
Cases per 1,000 inhabitants, 1939	0. 015
Cases per 1,000 inhabitants, estimated expectancy	0. 029
Deaths registered, 1939	694
Deaths per 1,000 inhabitants, 1939	0. 005
Cases reported for each death registered, 1939	2. 839
47 States:[1]	
Cases reported, 1939 (population 130,275,000)	[2] 1, 991
Deaths registered, 1939	715
Deaths per 1,000 inhabitants, 1939	0. 005
48 States:[1]	
Cases reported, 1939	[2] 1, 993

MUMPS (44C)

40 States:	
Cases reported, 1939 (population 98,306,000)	129, 714
Estimated expectancy based on years 1932-38	115, 385
Cases per 1,000 inhabitants, 1939	1. 319
Cases per 1,000 inhabitants, estimated expectancy	1. 198
Deaths registered, 1939	70
Deaths per 1,000 inhabitants, 1939	0. 001
Cases reported for each death registered, 1939	1, 853
44 States:	
Cases reported, 1939 (population 109,348,000)	[2] 129,731
Deaths registered, 1939	87
Deaths per 1,000 inhabitants, 1939	0. 001
47 States:	
Cases reported, 1939	[2] 131,826

[1] The District of Columbia is also included but not counted as a State.
[2] Includes the number of deaths used as cases when no cases are reported or when the reported number of cases is less than the number of deaths.

PELLAGRA (69)

18 States:	
Cases reported, 1939 (population 48,811,000)	10,200
Cases per 1,000 inhabitants, 1939	0.209
Deaths registered, 1939	1,925
Deaths per 1,000 inhabitants, 1939	0.039
Cases reported for each death registered, 1939	5.299
39 States:[1]	
Cases reported, 1939 (population 123,216,000)	[2] 10,717
Deaths registered, 1939	2,442
Deaths per 1,000 inhabitants, 1939	0.020
47 States:[1]	
Deaths registered, 1939 (population 130,275,000)	2,442
Deaths per 1,000 inhabitants, 1939	0.019

PNEUMONIA (ALL FORMS) (107-109)

20 States:[1]	
Cases reported, 1939 (population 89,689,000)	121,257
Cases per 1,000 inhabitants, 1939	1.352
Deaths registered, 1939	52,554
Deaths per 1,000 inhabitants, 1939	0.586
Cases reported for each death registered, 1939	2.307
47 States:[1]	
Deaths registered, 1939 (population 130,275,000)	77,602
Deaths per 1,000 inhabitants, 1939	0.596
48 States:[1]	
Cases reported, 1939	[2] [3] 147,658

POLIOMYELITIS (36)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	7,339
Estimated expectancy based on years 1932-38	3,726
Cases per 1,000 inhabitants, 1939	0.056
Cases per 1,000 inhabitants, estimated expectancy	0.029
Deaths registered, 1939	756
Deaths per 1,000 inhabitants, 1939	0.006
Cases reported for each death registered, 1939	9.708
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	7,343
Cases per 1,000 inhabitants, 1939	0.056

SCARLET FEVER (8)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	162,735
Estimated expectancy based on years 1932-38	207,103
Cases per 1,000 inhabitants, 1939	1.249
Cases per 1,000 inhabitants, estimated expectancy	1.630
Deaths registered, 1939	855
Deaths per 1,000 inhabitants, 1939	0.007
Cases reported for each death registered, 1939	190
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	162,897
Cases per 1,000 inhabitants, 1939	1.246

SEPTIC SORE THROAT (115b)

33 States:	
Cases reported, 1939 (population 85,360,000)	8,538
Cases per 1,000 inhabitants, 1939	0.100
Deaths registered, 1939	1,262
Deaths per 1,000 inhabitants, 1939	0.015
Cases reported for each death registered, 1939	6.765
43 States:[1]	
Cases reported, 1939 (population 119,201,000)	[2] 9,227
Deaths registered, 1939	1,951
Deaths per 1,000 inhabitants, 1939	0.016
46 States:[1]	
Cases reported, 1939	[2] 10,758

SMALLPOX (34)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	9,877
Estimated expectancy, based on years 1932-38	7,093
Cases per 1,000 inhabitants, 1939	0.076
Cases per 1,000 inhabitants, estimated expectancy	0.056
Deaths registered, 1939	39
Deaths per 1,000 inhabitants, 1939	0.0002
Cases reported for each death registered, 1939	253
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	9,877
Cases per 1,000 inhabitants, 1939	0.076

[1] The District of Columbia is also included but not counted as a State.

[2] Includes the number of deaths used as cases when no cases are reported or when the reported number of cases is less than the number of deaths.

[3] Includes 7,484 cases of lobar pneumonia only.

SYPHILIS (30)

48 States:[1]	
Cases reported, 1939 (population 130,763,000)	485,065
Cases per 1,000 inhabitants, 1939	3.709

TUBERCULOSIS (ALL FORMS) (13-22)

37 States:[1]	
Cases reported, 1939 (population 103,700,000)	92,292
Cases per 1,000 inhabitants, 1939	0.890
Deaths registered, 1939	47,828
Deaths per 1,000 inhabitants, 1939	0.461
Cases reported for each death registered, 1939	1.930
45 States:[1]	
Cases reported, 1939 (population 125,291,000)	[2]102,776
Deaths registered, 1939	58,312
Deaths per 1,000 inhabitants, 1939	0.465
47 States:[1]	
Deaths registered, 1939 (populaton 130,275,000)	61,319
Deaths per 1,000 inhabitants, 1939	0.471

TUBERCULOSIS (RESPIRATORY SYSTEM) (13)

21 States:[1]	
Cases reported, 1939 (population 63,639,000)	52,885
Cases per 1,000 inhabitants, 1939	0.831
Deaths registered, 1939	27,375
Deaths per 1,000 inhabitants, 1939	0.430
Cases reported for each death registered, 1939	1.932
46 States:[1]	
Cases reported, 1939 (population 129,632,000)	[2]81,451
Deaths registered, 1939	55,941
Deaths per 1,000 inhabitants, 1939	0.432
48 States:[1]	
Cases reported, 1939	[2]81,996

TYPHOID FEVER (1) AND PARATYPHOID FEVER (2)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	13,055
Estimated expectancy based on years 1932-38	18,679
Cases per 1,000 inhabitants, 1939	0.100
Cases per 1,000 inhabitants, estimated expectancy	0.147
Deaths registered, 1939	1,997
Deaths per 1,000 inhabitants, 1939	0.015
Cases reported for each death registered, 1939	6.537
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	13,069
Cases per 1,000 inhabitants, 1939	0.100

WHOOPING COUGH (9)

47 States:[1]	
Cases reported, 1939 (population 130,275,000)	183,046
Estimated expectancy based on years 1932-38	199,896
Cases per 1,000 inhabitants, 1939	1.405
Cases per 1,000 inhabitants, estimated expectancy	1.574
Deaths registered, 1939	3,008
Deaths per 1,000 inhabitants, 1939	0.023
Cases reported for each death registered, 1939	61
48 States:[1]	
Cases reported, 1939 (population 130,763,000)	183,188
Cases per 1,000 inhabitants, 1939	1.401

[1] The District of Columbia is also included but not counted as a State.

[2] Includes the number of deaths used as cases when no cases are reported, or when the reported number of cases is less than the number of deaths.

Cases reported, 1939, by months

Disease	Number of States [1]	January	February	March	April	May	June	July	August	September	October	November	December	Total
Anthrax in man (7)	12	6	4	4	6	4	2	4	9	3	2	7	6	57
Chickenpox (38e)	48	41,096	35,932	35,890	27,711	24,940	15,087	5,020	2,237	2,470	9,424	24,021	34,877	[2] 258,746
Dengue (38f)	8	7	9	15	12	2	5	10	30	31	9	8	6	144
Diphtheria (10)	48	2,476	1,961	1,781	1,489	1,226	961	1,051	1,401	2,330	3,221	3,396	2,748	[2] 24,053
Dysentery (amoebic) (27b)	39	197	210	199	277	246	376	307	331	266	261	207	162	[3] 3,039
Dysentery (bacillary) (27a)	41	510	472	508	775	2,236	4,641	4,445	2,233	2,327	1,230	1,173	771	[2] 21,327
Dysentery (unspecified) (27c)	5	38	36	50	46	196	185	103	191	113	106	69	50	1,183
Encephalitis, epidemic or lethargic (37)	47	90	58	75	60	66	49	86	158	129	112	99	80	[2, 3] 1,069
Influenza (33)	48	20,723	39,090	94,136	45,609	11,134	3,615	2,433	2,625	3,541	6,046	11,167	37,206	[2, 3] 277,616
Malaria (28)	40	1,948	1,613	2,187	3,764	5,613	9,377	14,079	14,103	14,000	8,847	4,841	2,283	[3] 82,655
Measles (35)	48	42,794	61,082	76,170	68,003	72,371	39,381	12,595	3,275	2,128	4,289	8,322	12,896	[2] 403,317
Meningitis, meningococcus (6)	48	234	227	229	192	171	129	127	100	116	138	146	140	[2, 3] 1,993
Mumps (44c)	47	16,377	18,567	24,267	19,226	17,250	9,455	4,066	2,550	2,326	3,468	6,329	7,945	[3] 131,826
Pellagra (69)	39	623	670	745	1,060	1,240	1,382	1,329	1,035	766	718	574	575	[3] 10,717
Pneumonia (all forms) (107–109)	48	21,227	22,206	23,271	17,581	11,690	6,336	4,454	3,996	4,375	6,257	9,536	15,437	[2, 3, 4] 147,658
Poliomyelitis (36)	48	74	71	45	97	184	278	643	1,602	2,018	1,366	669	295	[2] 7,343
Rabies in animals	29	594	520	539	449	452	400	265	294	252	228	221	204	[5] 4,418
Rabies in man (deaths) (38b)	47	6	2	3	2	1	8	6	3	4	2	4	2	[2] 44
Rocky Mountain spotted fever (39c)	32	3	2	4	41	103	134	106	109	36	13	5	4	[3] 560
Scarlet fever (8)	48	22,433	22,715	22,848	18,669	15,937	7,655	3,693	3,382	5,104	9,538	14,139	16,782	[2] 162,897
Septic sore throat (115b)	46	1,067	1,159	1,290	1,067	1,138	794	543	565	613	575	946	1,001	[3] 10,758
Smallpox (34)	48	1,658	1,677	1,470	1,555	1,321	845	368	115	112	113	234	409	9,877
Tuberculosis (all forms) (13–22)	45	7,902	7,387	9,672	9,135	9,347	9,520	9,206	9,168	8,188	8,343	7,515	7,376	[2, 3] 102,776
Tuberculosis (respiratory system) (13)	48	6,537	5,962	8,132	7,272	7,685	7,362	6,861	6,913	6,457	6,606	6,182	6,011	[2, 3] 81,996
Tularaemia (26a)	41	304	118	102	79	79	78	76	91	95	85	354	830	2,291
Typhoid and paratyphoid fever (1–2)	48	449	470	523	490	640	1,089	2,088	2,260	2,220	1,217	835	787	[2] 13,069
Typhus fever (39a) (39b)	21	188	120	85	109	175	188	359	484	432	317	275	266	[3] 2,998
Undulant fever (5)	47	235	224	235	277	293	347	358	402	348	219	270	293	3,501
Venereal diseases:														
Gonorrhea (25)	48	13,414	13,331	14,598	14,327	14,877	15,229	15,820	17,549	16,356	15,695	14,323	12,824	178,343
Syphilis (30)	48	37,831	42,622	43,102	43,386	45,363	40,275	40,702	41,551	40,001	38,073	38,902	33,257	485,065
Whooping cough (9)	48	19,741	17,771	19,114	16,333	17,512	17,154	17,815	13,592	10,805	9,869	11,927	11,550	[2] 183,188

[1] The District of Columbia is also included but not counted as a State.

[2] The following numbers of cases of certain diseases are not distributed by months but are included in the totals of the above table: Chickenpox, 41; diphtheria, 12; dysentery (bacillary), 6; encephalitis, epidemic or lethargic, 7; influenza, 291; measles, 11; meningitis, meningococcus, 44; pneumonia (all forms), 1,292; poliomyelitis, 1; rabies in man, 1; scarlet fever, 2; tuberculosis (all forms), 17; tuberculosis (respiratory system), 16; typhoid fever, 1; whooping cough, 5.

[3] Includes the number of deaths used as cases when no cases are reported, or when the reported number of cases is less than the number of deaths.

[4] Includes 4,460 cases of lobar pneumonia only in Massachusetts, and 3,024 cases of lobar pneumonia only in California.

[5] Exclusive of New York City.

NOTE.—Figures in parentheses are disease title numbers from the International List of Causes of Death, 1938.

Deaths registered, 1939, by months

Disease	Number of States [1]	January	February	March	April	May	June	July	August	September	October	November	December	Total
Anthrax in man (7)	46				2		1	1	4	1				9
Chickenpox (38e)	47	18	14	18	15	7	11	7		1	1	3	14	[2] 110
Dengue (38f)	1								1					1
Diphtheria (10)	47	245	164	151	101	82	60	87	101	161	259	306	248	[2] 2,022
Dysentery (amoebic) (27b)	47	13	15	20	18	17	30	26	25	33	22	18	20	[2] 282
Dysentery (bacillary) (27a)	46	29	18	27	27	65	155	179	150	99	113	66	29	[2] 1,046
Dysentery (unspecified) (27c)	3	11	6	9	25	50	72	51	39	24	27	13	18	345
Encephalitis, epidemic or lethargic (37)	47	64	54	54	62	51	33	54	67	66	47	36	50	[2] 645
Influenza (33)	47	2,546	3,039	5,703	3,156	1,574	529	356	302	374	611	1,013	1,802	[2] 21,834
Malaria (28)	47	52	37	56	54	96	115	242	215	263	200	131	68	[2] 1,750
Measles (35)	47	132	179	180	182	186	83	50	17	15	15	16	32	[2] 1,171
Meningitis, meningococcus (6)	47	109	85	59	59	52	43	59	38	42	45	55	47	[2] 715
Mumps (44c)	45	10	8	8	9	10	8	8	6	4	2	6	5	[2] 87
Pellagra (69)	47	191	167	198	193	186	215	218	164	170	178	184	179	[2] 2,442
Pneumonia (all forms) (107–109)	47	11,987	10,947	11,455	7,633	5,497	3,218	2,909	2,695	3,143	4,092	5,423	7,325	[2] 77,602
Poliomyelitis (36)	47	26	25	16	27	37	46	70	128	142	113	68	42	[2] 756
Rabies in man (38b)	47	6	2	3	2	1	8	6	3	4	2	4	2	[2] 44
Rocky Mountain spotted fever (39c)	46			1	6	18	24	19	16	6		1	3	94
Scarlet fever (8)	47	115	105	141	97	60	49	33	26	34	46	60	88	[2] 855
Septic sore throat (115b)	43	182	187	151	187	167	153	140	159	126	161	149	189	1,951
Smallpox (34)	47	5	9	5	6	7	3	2				2		39
Tuberculosis (all forms) (13–22)	47	5,136	5,018	5,801	5,483	5,552	5,034	4,949	4,817	4,482	4,547	4,611	4,792	[2] 61,319
Tuberculosis (respiratory system) (13)	46	4,701	4,604	5,306	4,983	5,017	4,535	4,476	4,376	4,091	4,152	4,261	4,412	[2] 55,941
Tularaemia (26a)	47	37	13	19	8	5	9	11	9	6	8	40	90	[2] 258
Typhoid and paratyphoid fever (1–2)	47	102	87	97	83	90	172	253	332	311	186	139	97	[2] 1,997
Typhus fever (39a, b)	46	11	6	6	5	5	14	13	21	24	10	12	16	143
Undulant fever (5)	47	4	10	5	4	10	11	12	18	17	10	13	6	[2] 121
Whooping cough (9)	47	252	251	342	264	265	277	260	223	192	203	159	162	[2] 3,008

[1] The District of Columbia is also included but not counted as a State.

[2] The following numbers of deaths from certain diseases are not distributed by months but are included in the totals of the above table: Chickenpox, 1; diphtheria, 57; dysentery (amoebic), 25; dysentery (bacillary), 89; encephalitis, epidemic or lethargic, 7; influenza, 829; malaria, 221; measles, 84; meningitis, meningococcus, 22; mumps, 3; pellagra, 199; pneumonia (all forms), 1,278; poliomyelitis, 16; rabies in man, 1; scarlet fever, 1; tuberculosis (all forms), 1,097; tuberculosis (respiratory system), 1,027; tularaemia, 3; typhoid fever, and paratyphoid fever, 48; undulant fever, 1; whooping cough, 158.

NOTE.—Figures in parentheses are disease title numbers from the International List of Causes of Death, 1938.

COURT DECISION ON PUBLIC HEALTH

Trichinosis held compensable under workmen's compensation act.—(Massachusetts Supreme Judicial Court; *Destefano* v. *Alpha Lunch Co. of Boston, Vaida* v. *Same*, 30 N.E.2d 827; decided January 3, 1941.) In actions for breach of the implied warranty of fitness of food under a Massachusetts statute it appeared that the plaintiffs worked for the defendant company in one of its restaurants. Each plaintiff took two meals a day, except Sunday, at the restaurant, the meals forming part of the pay. Both plaintiffs became ill with trichinosis and testified that, during the 2 weeks preceding the onset of the disease, they ate pork and other products of the pig at the defendant's restaurant and nowhere else. The defendant was insured under the workmen's compensation act and the plaintiffs had made no reservation of common law rights under that act.

The supreme court took the view that what happened to the plaintiffs constituted a "personal injury" within the workmen's compensation act. "It differed from the inhalation of germs of disease, illustrated by *Smith's Case, Mass.*, 30 N.E.2d 536.[1] It resembled more the cases of poisoning therein cited, and *Osterbrink's Case*, 229 Mass. 407, 118 N.E. 657, where the employee drank muriatic acid by mistake for water." Also the court was of the opinion that such personal injury arose out of and in the course of the plaintiffs' employment.

"Since the injury was compensable under the workmen's compensation act, it will not support an action against the employer at law, whether in tort or in contract, or whether or not based upon a statute. * * *"

The action of the court below in ordering judgment for the defendant was sustained.

DEATHS DURING WEEK ENDED FEBRUARY 1, 1941

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended Feb. 1, 1941	Corresponding week, 1940
Data from 88 large cities of the United States:		
Total deaths	10,112	10,162
Average for 3 prior years	9,586	
Total deaths, first 5 weeks of year	49,361	48,141
Deaths under 1 year of age	558	577
Average for 3 prior years	557	
Deaths under 1 year of age, first 5 weeks of year	2,816	2,766
Data from industrial insurance companies:		
Policies in force	64,727,301	66,327,780
Number of death claims	14,799	13,817
Death claims per 1,000 policies in force, annual rate	11.9	10.9
Death claims per 1,000 policies, first 5 weeks of year, annual rate	10.6	10.4

[1] See Public Health Reports, January 31, 1941, p. 197.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED FEBRUARY 8, 1941

Summary

For the third successive week the incidence of influenza has recorded a decrease, with a total of 38,241 cases reported by the State health officers, as compared with 72,578 cases for the preceding week. The decline is noted for all geographic areas except the Pacific, where California reported 1,387 cases, as compared with 1,149 last week. It appears likely, however, that this increase may be attributed to delayed reports. West Virginia, with 6,046 cases; Virginia, with 5,976; and Texas, with 4,580, reported the highest incidence for the current week, although a sharp decline from the preceding week occurred in each of these States.

Of the other eight communicable diseases, only measles, poliomyelitis, and whooping cough were above the 5-year (1936–40) median expectancy. Also, the cumulative totals of these diseases for the first 6 weeks of the current year were above the cumulative medians. The number of cases of measles reported for the current week is approximately two and one-half times the 5-year median, while whooping cough was only slightly above the expectancy. The 29 cases of poliomyelitis (as compared with a 5-year median of 18) exceed the number reported for the corresponding week in any of the preceding 5 years. The cases were scattered, with only three States reporting as many as 3 cases.

Of 58 cases of smallpox, 25 cases were reported in the East North Central States (12 in Wisconsin and 8 in Michigan). One case of tularemia each was reported in Maryland, North Carolina, and South Carolina; and of 24 cases of endemic typhus fever, 14 were reported in Georgia.

For the current week the Bureau of the Census reports 10,214 deaths in 88 major cities of the United States, as compared with 10,112 for the preceding week and a 3-year (1938–40) average of 9,525 for the corresponding week. The current figure is 689 above the 3-year average as compared with a similar excess of 526 for the preceding week.

Telegraphic morbidity reports from State health officers for the week ended February 8, 1941, and comparison with corresponding week of 1940 and 5-year median

In these tables a zero indicates a definite report, while leader simply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended—		Median 1936–40	Week ended—		Median 1936–40	Week ended—		Median 1936–40	Week ended—		Median 1936–40
	Feb. 8, 1941	Feb. 10, 1940		Feb. 8, 1941	Feb. 10, 1940		Feb. 8, 1941	Feb. 10, 1940		Feb. 8, 1941	Feb. 10, 1940	
NEW ENG.												
Maine	0	1	2	63	1	3	70	[illegible]	155	0	0	0
New Hampshire	0	0	0	5	2	2	8	52	44	0	0	0
Vermont	0	0	0	26	------	------	10	3	27	0	0	0
Massachusetts	1	3	3	------	------	------	432	272	435	1	0	2
Rhode Island	0	1	1	10	------	------	0	111	99	0	0	0
Connecticut	0	0	1	317	2	4	30	177	177	0	0	0
MID. ATL.												
New York	11	22	34	[1] 427	[1] 36	[1] 50	3,086	267	673	1	0	4
New Jersey	15	8	11	1,156	29	29	844	66	66	2	0	1
Pennsylvania	25	24	44	------	------	------	2,919	80	170	2	7	7
E. NO. CEN.												
Ohio	10	15	20	863	22	20	1,836	22	24	3	1	3
Indiana	11	18	39	173	90	52	183	6	14	0	0	4
Illinois	19	30	32	195	134	134	1,831	30	36	1	0	4
Michigan [2]	2	9	12	155	11	3	1,320	251	251	0	0	1
Wisconsin	0	4	3	715	77	65	585	182	182	1	0	1
W. NO. CEN.												
Minnesota	3	3	3	698	1	1	5	359	120	2	0	1
Iowa	13	3	6	396	25	8	130	97	55	0	1	1
Missouri	4	10	10	68	33	162	74	5	10	1	0	1
North Dakota	0	3	2	84	61	15	11	13	13	0	0	0
South Dakota	1	1	1	22	4	4	18	7	4	0	0	0
Nebraska	1	0	3	14	2	2	6	31	22	0	0	0
Kansas	4	10	11	340	101	68	174	301	20	3	1	1
SO. ATL.												
Delaware	1	0	0	10	------	------	50	1	24	0	0	0
Maryland [2]	5	7	9	351	263	103	61	4	112	0	2	2
Dist. of Col	0	0	6	79	19	5	14	2	11	0	1	1
Virginia	6	12	22	5,976	2,662	------	498	42	99	1	2	10
West Virginia [2]	2	11	11	6,046	460	151	125	15	15	0	4	3
North Carolina	16	25	24	599	121	67	182	107	107	0	1	2
South Carolina [3]	7	3	3	3,060	1,331	1,009	47	6	23	2	2	2
Georgia [3]	7	2	11	1,509	728	490	202	76	76	2	0	1
Florida	4	4	9	387	50	4	21	41	41	0	0	0
E. SO. CEN.												
Kentucky	11	10	9	246	86	86	203	35	70	3	0	6
Tennessee	10	9	13	2,003	424	176	99	54	64	3	2	4
Alabama [3]	5	5	8	3,561	536	334	476	73	73	5	2	2
Mississippi [2]	2	4	6	------	------	------	------	------	------	3	2	1
W. SO. CEN.												
Arkansas	12	8	8	767	1,698	235	83	4	4	0	0	1
Louisiana [3]	5	11	11	218	360	44	7	15	15	2	0	0
Oklahoma	15	8	8	657	664	285	11	4	15	0	1	1
Texas [3]	36	51	51	4,580	4,437	940	218	270	167	5	3	5
MOUNTAIN												
Montana	10	1	1	116	7	7	8	28	20	0	0	1
Idaho	2	1	1	------	6	6	25	163	88	0	1	0
Wyoming	0	2	1	189	4	------	14	4	5	0	0	0
Colorado	9	9	9	311	26	------	85	32	34	0	0	0
New Mexico	2	1	3	9	7	7	72	9	29	0	0	0
Arizona	2	2	4	281	297	175	80	13	13	0	0	0
Utah [2]	2	0	0	66	125	------	15	190	81	0	1	0
Nevada	0	------	------	------	------	------	0	------	------	0	------	------
PACIFIC												
Washington	0	3	2	52	35	4	70	676	182	0	0	1
Oregon	8	2	2	54	107	76	325	247	34	0	0	0
California	18	22	25	[4] 1,387	1,499	522	101	433	433	3	1	1
Total	317	378	491	38,241	16,583	4,577	16,664	5,085	6,519	46	35	89
6 weeks	1,840	2,628	3,574	494,449	82,180	20,877	70,927	25,982	31,671	264	198	552

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended February 8, 1941, and comparison with corresponding week of 1940 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40	Week ended		Median 1936–40
	Feb. 8, 1941	Feb. 10, 1940		Feb. 8, 1941	Feb. 10, 1940		Feb. 8, 1941	Feb. 10, 1940		Feb. 8, 1941	Feb. 10, 1940	
NEW ENG.												
Maine	0	0	0	9	19	25	0	0	0	0	0	0
New Hampshire	0	0	0	4	4	7	0	0	0	0	0	0
Vermont	0	0	0	4	9	16	0	0	0	0	0	0
Massachusetts	0	1	0	143	134	250	0	0	0	1	2	1
Rhode Island	0	0	0	10	12	30	0	0	0	0	0	1
Connecticut	0	0	0	43	90	97	0	0	0	1	3	1
MID. ATL.												
New York	0	2	1	380	665	690	0	0	0	5	6	5
New Jersey	0	0	0	309	333	172	0	0	0	0	2	1
Pennsylvania	1	0	0	248	370	472	0	0	0	0	8	8
E. NO. CEN.												
Ohio	0	1	1	296	277	313	5	0	3	1	0	1
Indiana	0	0	0	145	221	221	0	6	6	4	1	1
Illinois	2	0	0	454	583	622	0	2	11	3	1	2
Michigan [2]	1	1	1	142	261	497	8	2	3	0	0	4
Wisconsin	8	1	0	165	160	298	12	5	5	0	0	1
W. NO. CEN.												
Minnesota	1	0	0	49	112	150	6	5	8	0	0	0
Iowa	2	4	0	46	70	182	3	9	33	0	0	1
Missouri	3	0	0	76	91	145	3	2	17	0	1	1
North Dakota	0	0	0	16	28	28	0	0	2	0	0	1
South Dakota	0	0	0	17	39	30	0	4	6	1	0	0
Nebraska	0	0	0	25	20	53	0	0	5	0	0	0
Kansas	0	0	0	72	75	209	0	1	10	0	0	0
SO. ATL.												
Delaware	0	0	0	7	10	7	0	0	0	0	0	0
Maryland [3]	1	0	0	82	62	62	0	0	0	2	2	1
Dist. of Col	0	0	0	9	21	18	0	0	0	1	1	0
Virginia	0	0	0	47	28	40	0	0	0	4	1	4
West Virginia [3]	2	0	0	30	77	50	0	0	0	2	0	2
North Carolina	3	0	0	48	53	53	0	0	0	2	3	3
South Carolina [3]	0	0	0	6	3	3	0	0	0	0	3	3
Georgia [3]	0	2	0	21	25	19	0	2	0	0	2	3
Florida	2	0	0	2	11	10	0	0	0	0	3	2
E. SO. CEN.												
Kentucky	0	0	1	83	94	68	0	0	0	4	3	3
Tennessee	0	0	0	102	64	44	0	1	1	2	0	3
Alabama [3]	0	2	1	14	13	22	0	0	1	1	3	3
Mississippi [3]	1	2	1	5	3	8	1	0	0	0	1	1
W. SO. CEN.												
Arkansas	1	0	0	9	3	15	2	3	1	2	2	2
Louisiana [3]	0	0	0	4	13	13	2	0	0	3	2	5
Oklahoma	1	1	1	18	31	31	1	1	2	0	1	2
Texas [3]	1	1	1	75	75	89	5	1	5	10	3	3
MOUNTAIN												
Montana	0	0	0	25	53	53	1	0	11	1	0	1
Idaho	0	1	0	16	42	26	1	1	2	0	1	1
Wyoming	0	0	0	8	4	12	0	0	4	0	0	0
Colorado	0	0	0	37	59	59	6	13	13	0	0	0
New Mexico	1	0	0	4	13	25	0	0	0	3	3	3
Arizona	0	0	0	7	13	22	2	1	0	1	0	0
Utah [2]	1	0	0	7	31	31	0	0	0	0	0	0
Nevada	0	-----	-----	0	-----	-----	0	-----	-----	0	-----	-----
PACIFIC												
Washington	0	0	0	24	59	62	0	3	12	0	3	2
Oregon	0	0	0	18	22	45	0	0	2	1	1	1
California	2	2	2	105	140	200	0	1	11	5	10	4
Total	29	21	18	3,466	4,595	6,146	58	63	371	60	72	87
6 weeks	217	203	124	19,470	25,951	35,937	304	453	1,828	445	475	661

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended February 8, 1941, and comparison with corresponding week of 1940 and 5-year median—Con.

Division and State	Whooping cough, Week ended Feb. 8, 1941	Whooping cough, Week ended Feb. 10, 1940
NEW ENG.		
Maine	8	77
New Hampshire	2	4
Vermont	8	47
Massachusetts	272	144
Rhode Island	6	13
Connecticut	52	64
MID. ATL.		
New York	337	394
New Jersey	102	95
Pennsylvania	435	341
E. NO. CEN.		
Ohio	341	92
Indiana	9	46
Illinois	107	80
Michigan [2]	175	115
Wisconsin	150	93
W. NO. CEN.		
Minnesota	58	25
Iowa	29	10
Missouri	53	15
North Dakota	18	18
South Dakota	8	11
Nebraska	15	4
Kansas	70	55
SO. ATL.		
Delaware	8	9
Maryland [2]	94	165
Dist. of Col.	5	15
Virginia	232	57
West Virginia [2]	102	6
North Carolina	302	76
South Carolina [3]	61	6
SO. ATL.—continued		
Georgia [3]	15	38
Florida	17	18
E. SO. CEN.		
Kentucky	38	60
Tennessee	73	41
Alabama [3]	49	7
Mississippi [2]	----------	----------
W. SO. CEN.		
Arkansas	25	6
Louisiana [3]	1	9
Oklahoma	31	4
Texas [3]	343	118
MOUNTAIN		
Montana	7	1
Idaho	14	5
Wyoming	9	2
Colorado	59	20
New Mexico	13	37
Arizona	5	26
Utah [2]	74	552
Nevada	0	----------
PACIFIC		
Washington	123	19
Oregon	13	36
California	424	154
Total	4,392	3,230
6 weeks	25,434	16,720

[1] New York City only.
[2] Period ended earlier than Saturday.
[3] Typhus fever, week ended Feb. 8, 1941, 24 cases as follows: South Carolina, 5; Georgia, 14; Alabama, 2; Louisiana, 2; Texas, 1.
[4] Approximately 1,000 delayed reports for November and December included.

WEEKLY REPORTS FROM CITIES

City reports for week ended January 25, 1941

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities:											
5-year average	163	1,347	138	2,653	933	1,728	35	365	18	1,119	--------
Current week [1]	67	6,912	205	6,206	739	1,129	4	375	12	1,202	--------
Maine:											
Portland	0	1	1	1	6	4	0	0	0	13	36
New Hampshire:											
Concord	0	------	0	0	0	1	0	1	0	0	16
Manchester	0	------	2	0	1	13	0	0	0	0	15
Nashua	0	------	0	0	0	7	0	1	0	1	7
Vermont:											
Barre	------	------	------	------	------	------	------	------	------	------	------
Burlington	0	3	0	4	0	0	0	0	0	0	11
Rutland	0	------	0	0	0	0	0	0	0	0	9
Massachusetts:											
Boston	0	------	6	88	51	41	0	9	0	80	349
Fall River	0	------	1	0	2	3	0	1	0	4	51
Springfield	0	1	1	3	1	10	0	0	0	1	53
Worcester	0	------	0	52	6	11	0	3	1	0	78
Rhode Island:											
Pawtucket	0	------	0	0	0	0	0	0	0	0	19
Providence	0	12	3	1	11	0	0	2	0	5	101
Connecticut:											
Bridgeport	0	58	1	1	3	3	0	0	0	6	54
Hartford	0	80	0	1	5	1	0	1	0	1	54
New Haven	0	25	2	0	1	7	0	0	1	25	49
New York:											
Buffalo	0	20	5	57	13	21	0	5	0	16	165
New York	17	522	10	1,548	111	220	0	82	1	139	1,693
Rochester	0	------	0	6	4	3	0	0	0	8	74
Syracuse	0	------	0	0	3	3	0	0	0	3	44
New Jersey:											
Camden	0	15	3	24	2	4	0	3	0	2	47
Newark	0	41	0	122	17	20	0	10	0	7	137
Trenton	0	6	2	2	5	66	0	0	0	1	50
Pennsylvania:											
Philadelphia	1	50	7	773	50	100	0	28	0	70	701
Pittsburgh	0	66	8	1	29	5	0	9	0	40	226
Reading	1	------	0	229	5	0	0	1	0	15	33
Scranton	0	------	------	1	------	0	0	------	0	1	------
Ohio:											
Cincinnati	0	43	2	28	11	14	0	5	0	2	145
Cleveland	3	437	2	447	17	29	0	9	0	93	216
Columbus	1	6	6	10	6	8	0	3	0	9	104
Toledo	0	2	1	3	7	7	0	4	1	22	91
Indiana:											
Anderson	0	------	0	0	2	0	0	0	0	0	10
Fort Wayne	0	------	0	13	3	1	0	1	0	0	29
Indianapolis	2	------	2	6	20	19	0	3	0	15	131
Muncie	0	------	1	11	1	6	0	1	0	0	13
South Bend	0	------	0	2	2	0	0	0	0	0	23
Terre Haute	0	1	0	0	1	0	0	0	0	0	12
Illinois:											
Alton	0	1	1	0	1	4	0	0	0	0	9
Chicago	10	45	3	900	47	170	0	48	3	63	803
Elgin	0	------	0	3	4	1	0	0	0	0	21
Moline	0	------	0	4	0	0	0	0	0	0	15
Springfield	0	------	0	1	6	5	0	0	1	2	28
Michigan:											
Detroit	4	86	3	797	17	92	0	10	0	119	306
Flint	0	------	2	18	7	3	0	0	0	11	37
Grand Rapids	0	------	1	0	1	11	0	0	0	16	36
Wisconsin:											
Kenosha	0	------	0	8	0	1	0	0	0	0	9
Madison	0	------	0	2	0	1	0	0	0	1	10
Milwaukee	0	------	0	15	5	39	0	0	0	54	96
Racine	------	------	------	------	------	------	------	------	------	------	------
Superior	0	------	0	0	0	2	0	0	0	1	8

[1] Figures for Barre, Racine, and Boise estimated; reports not received.

City reports for week ended January 25, 1941—Continued

State and city	Diphtheria cases	Influenza Cases	Influenza Deaths	Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
Minnesota:											
Duluth	0	3	0	0	2	0	1	0	0	5	21
Minneapolis	0		1	909	1	8	0	2	0	13	112
St. Paul	1	2	2	2	5	6	0	4	0	5	69
Iowa:											
Cedar Rapids	0			1		4	0		0	0	
Davenport	0			0		2	0		0	1	
Des Moines	3		0	0	0	11	0	0	0	1	28
Sioux City	0			0		3	0		0	8	
Waterloo	0			1		2	0		0	4	
Missouri:											
Kansas City	0	1	5	3	12	12	3	5	0	6	115
St. Joseph	0		0	0	9	0	0	0	0	2	27
St. Louis	1	31	9	7	26	19	0	7	1	14	270
North Dakota:											
Fargo	0		0	0	0	0	0	0	0	7	4
Grand Forks	0			0		0	0		0	0	
Minot	0			1		0	0		0	0	
South Dakota:											
Aberdeen	0			0		2	0		0	0	
Sioux Falls	0		0	0	0	1	0	0	0	0	7
Nebraska:											
Lincoln	1			0		1	0		0	1	
Omaha	0		1	0	10	8	0	1	0	0	65
Kansas:											
Lawrence	0	27	0	1	0	0	0	0	0	0	3
Topeka	0		0	20	0	1	0	0	0	0	11
Wichita	1	3	1	2	5	2	0	1	0	15	32
Delaware:											
Wilmington	0		0	4	6	1	0	0	0	7	42
Maryland:											
Baltimore	2	105	5	5	16	35	0	10	0	60	270
Cumberland	0	1	0	2	0	0	0	0	0	0	9
Frederick	0		0	0	0	0	0	0	0	0	3
Dist. of Col.:											
Washington	1	168	2	5	14	11	0	9	0	7	171
Virginia:											
Lynchburg	0		0	2	1	1	0	0	0	0	15
Norfolk	0	260	1	5	5	1	0	3	0	3	37
Richmond	0		7	2	4	2	0	3	0	0	77
Roanoke	0		0	26	4	1	0	0	0	25	26
West Virginia:											
Charleston	0	19	1	5	1	1	0	0	0	0	10
Huntington	0			0		0	0		0	0	
Wheeling	0		2	0	2	2	0	0	0	8	29
North Carolina:											
Gastonia	0	3		0		0	0		0	1	
Raleigh	0	15	0	0	2	1	0	0	0	6	7
Wilmington	0		0	0	4	0	0	0	0	0	15
Winston-Salem	3	18	0	1	0	1	0	2	0	27	27
South Carolina:											
Charleston	0	2,274	4	10	7	1	0	2	0	0	33
Florence	1	322	0	19	0	0	0	1	0	0	10
Greenville	0		0	2	7	0	0	1	0	3	20
Georgia:											
Atlanta	0	693	12	1	7	6	0	10	1	1	112
Brunswick	0	1	1	0	1	0	0	0	0	0	5
Savannah	0	256	7	0	5	0	0	2	0	0	51
Florida:											
Miami	0	46	2	0	3	1	0	0	0	1	63
Tampa	2	3	1	0	2	1	0	2	0	0	36
Kentucky:											
Ashland	0	6	2	0	0	1	0	1	0	1	11
Covington	1	2	0	3	3	2	0	1	0	0	22
Lexington	0		3	20	3	1	0	2	0	2	22
Louisville	0	93	1	21	9	17	0	2	0	10	66
Tennessee:											
Knoxville	0	519	4	1	9	4	0	2	0	0	58
Memphis	0	147	11	8	6	4	0	5	0	13	98
Nashville	0		6	4	7	6	0	1	1	12	73
Alabama:											
Birmingham	1	673	8	5	9	0	0	1	0	1	96
Mobile	1	22	7	0	5	0	0	2	0	0	35
Montgomery	0	87		2		4	0		0	0	

City reports for week ended January 25, 1941—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Arkansas:											
Fort Smith	0	3	------	0	------	0	0	------	0	4	------
Little Rock	0	116	1	2	11	0	0	2	0	2	58
Louisiana:											
Lake Charles	0	------	0	1	1	0	0	0	0	0	7
New Orleans	2	61	5	0	24	8	0	11	2	5	187
Shreveport	0	------	3	0	3	0	0	3	0	0	42
Oklahoma:											
Oklahoma City	0	------	1	4	4	3	0	0	0	2	54
Tulsa	2	------	1	0	6	0	1	2	0	4	37
Texas:											
Dallas	0	7	7	2	5	6	0	4	0	0	85
Fort Worth	0	------	3	36	4	2	0	1	0	0	43
Galveston	1	57	0	0	1	0	0	0	0	1	16
Houston	1	106	4	1	8	2	0	6	0	0	105
San Antonio	1	32	8	0	7	2	0	7	0	5	76
Montana:											
Billings	0	------	0	0	1	2	0	1	0	0	11
Great Falls	0	------	0	0	2	7	0	0	0	0	10
Helena	0	98	0	0	1	0	0	1	0	0	12
Missoula	0	97	1	1	0	0	0	0	0	0	6
Idaho:											
Boise	------	------	------	------	------	------	------	------	------	------	------
Colorado:											
Colorado Springs	0	------	0	2	2	2	0	1	0	8	10
Denver	1	65	3	6	9	8	0	3	0	11	109
Pueblo	0	------	0	0	1	1	0	1	0	2	11
New Mexico:											
Albuquerque	0	1	0	4	1	0	0	3	0	0	15
Utah:											
Salt Lake City	2	------	0	7	4	2	0	0	0	9	35
Washington:											
Seattle	0	110	1	5	4	3	0	5	0	8	104
Spokane	0	1	2	0	0	2	0	1	0	2	45
Tacoma	0	------	0	1	2	1	0	1	0	10	45
Oregon:											
Portland	0	6	1	18	2	4	0	3	0	0	90
Salem	0	6	------	1	------	0	0	------	0	2	------
California:											
Los Angeles	3	173	2	4	5	27	0	17	0	48	442
Sacramento	4	9	3	0	2	7	0	3	0	1	42
San Francisco	0	30	1	0	6	1	0	6	0	41	197

State and city	Meningitis, meningococcus		Poliomyelitis cases	State and city	Meningitis, meningococcus		Poliomyelitis cases
	Cases	Deaths			Cases	Deaths	
New York:				South Carolina:			
Buffalo	1	0	0	Greenville	1	0	0
New York	2	1	1	Florida:			
New Jersey:				Miami	0	0	2
Newark	1	0	0	Alabama:			
Pennsylvania:				Birmingham	1	1	0
Philadelphia	2	1	0	Louisiana:			
Ohio:				Shreveport	0	1	0
Cincinnati	0	0	1	Texas:			
Cleveland	1	0	0	Dallas	0	0	1
Indiana:				Galveston	1	0	0
Indianapolis	1	1	0	Oregon:			
Illinois:				Portland	1	0	0
Chicago	0	0	2	California:			
Wisconsin:				Los Angeles	1	0	1
Milwaukee	1	0	0				

Dengue fever.—Cases: Charleston, S. C., 3.
Encephalitis, epidemic or lethargic.—Cases: New York, 6.
Pellagra.—Cases: Charleston, S. C., 2; Atlanta, 1; Savannah, 1.
Rabies in man.—Deaths: San Francisco, 1.
Typhus fever.—Cases: New York, 3; Charleston, S. C., 1; Atlanta, 1; Miami, 1; Tampa, 1.

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FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended January 4, 1941.—During the week ended January 4, 1941, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis	2	13	3	4	18				5	45
Chickenpox		2	2	78	311	34	10	59	87	583
Diphtheria		54	3	15		7	1			80
Dysentery				2						2
Influenza		254	16		438	6			826	1,540
Lethargic encephalitis		1								1
Measles		147	11	21	490	71	32	128	195	1,095
Mumps				47	65	16	1	12	8	149
Pneumonia		6			40	3			17	66
Poliomyelitis				1						1
Scarlet fever	4	30	1	53	137	8		19	9	261
Tuberculosis		13	5	17	40	2		1		78
Typhoid and paratyphoid fever				8					1	9
Whooping cough			19	49	134	5	1	2	10	220

CUBA

Provinces—Notifiable diseases—4 weeks ended December 7, 1940.—During the 4 weeks ended December 7, 1940, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer		1		6		8	15
Chickenpox		2				1	3
Diphtheria		26		1	2	7	36
Hookworm disease				1			1
Leprosy				1			1
Malaria	67	15	2	7	2	72	165
Measles			2				2
Poliomyelitis		2					2
Scarlet fever				2			2
Trachoma				3			3
Tuberculosis	28	32	12	27	16	28	143
Typhoid fever	31	79	7	22	13	17	169

JAMAICA

Communicable diseases—4 weeks ended January 18, 1941.—During the 4 weeks ended January 18, 1941, cases of certain communicable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Chickenpox	1	11	Poliomyelitis		1
Diphtheria	2	4	Puerperal sepsis		6
Dysentery	8	8	Tuberculosis	24	83
Leprosy	1		Typhoid fever	5	36

VENEZUELA

Caracas—Poliomyelitis.—An increase in the number of poliomyelitis cases has been reported in Caracas, Venezuela (population 204,000), with 9 cases in November 1940 and 36 cases from December 1, 1940, to January 11, 1941, as compared with 6 cases from January to October (inclusive) 1940. The disease was stated to be mild, with only 4 deaths reported.

YUGOSLAVIA

Notifiable diseases—4 weeks ended December 1, 1940.—During the 4 weeks ended December 1, 1940, certain notifiable diseases were reported in Yugoslavia as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	15	1	Paratyphoid fever	16	
Cerebrospinal meningitis	59	14	Poliomyelitis	8	
Diphtheria and croup	730	34	Scarlet fever	393	1
Dysentery	682	91	Sepsis	7	3
Erysipelas	194	3	Tetanus	25	8
Favus	4		Typhoid fever	458	33
Leprosy	1		Typhus fever	23	1
Lethargic encephalitis	1				

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—A cumulative table giving current information regarding the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS of January 31, 1941, pages 206-210. A similar table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Smallpox

Japan.—According to a report dated January 23, 1941, an outbreak of smallpox has been reported in Japan. In Aomori Prefecture new cases increased to 36 between January 1 and 21, 1941. For the same period Akita Prefecture reported 5 cases and Tokyo Prefecture 5 cases. Three deaths had occurred.

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